THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 76 OCTOBER, 1956 No. 4

SIXTY YEARS OF CARDIOVASCULAR ROENTGENOLOGY

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IN OCTOBER, 1906 when this JOURNAL made its first appearance as the AMERICAN QUARTERLY OF ROENTGENOLOGY, cardiovascular roentgenology was still in its infancy, even though in the eleven years which had followed Röntgen's startling announcement, some rather significant progress had been made.

1896–1906

Less than one year following the discovery of the X-ray, Dr. Francis H. Williams of Boston published two papers concerning the use of roentgenology in cardiac diagnosis. The first, entitled *Notes on X-rays in Medicine* was read before the Association of American Physicians on April 30, 1896. He reported that "the first medical case I examined was that of a man with an enlarged heart (seven inches in transverse diameter). I found that the outline of the heart as seen from the front of the body through the fluorescope, corresponded in a general way to the outline drawn on the skin with percussion as a guide. It was interesting to note that the heart could be made out through the man's waistcoat and two shirts." His second paper was published in the Boston Medical and Surgical Journal of October 1, 1896 and was entitled *A Method for More Fully Determining the Outline of the Heart by Means of the Fluorescope.* In this presentation, Dr. Williams reported, among other things, that "the fluorescope also reveals the presence of certain aneurisms if their outline encroaches upon the visible portion of the comparatively transparent lung tissue; likewise, in connection with other physical signs, the presence of fluid in the pleural and pericardial sacs, the presence of pneumonia, edema of the lungs, infarctions, tuberculosis, hydro-thorax, pneumo-thorax, etc."

Dr. Williams had graduated at the age of twenty-one from the Massachusetts Institute of Technology and had received his degree of Doctor of Medicine from Harvard University four years later. As a Visiting Physician to the Boston City Hospital he had begun his work with the roentgen-rays. Initially because of the lack of hospital facilities (a common deterrent in that era) he had been compelled to transport his patients to the Rogers Laboratory of Physics at the Massachusetts Institute of Technology where a fluoroscope was available. Eventually the trustees of the Boston City Hospital made available to him a small basement room

which was his "X-ray Department" for nineteen years. During this period his particular interest was diseases of the cardiorespiratory system. By the end of 1898, Dr. Williams had studied, mainly by roentgenoscopy, virtually all cardiac diseases recognized at that time and had described quite accurately the roentgen features of pericardial effusion, intrathoracic aneurysm, and cardiac hypertrophy. The first edition of his classic text *The Roentgen Rays in Medicine and Surgery* contained eighty-four information-packed pages on the heart and great vessels. This remarkable volume is still worthwhile reading for the radiologist.

The superiority of roentgenoscopy for the determination of cardiac size and contour over the older percussion method became more definitely established in 1902 when Friedrich H. L. Moritz of Germany introduced orthodiagraphy. It is difficult for a young radiologist to visualize the widespread acceptance which this method received, and it is doubtful if many young radiologists have seen orthodiagraphy performed. It consisted of tracing the shadows of the margins of the heart and great vessels on a thin sheet of transparent paper which was affixed to the fluoroscopic screen. An accurate delineation of the cardiac border was assured by the use of the thin slit screen and distortion was practically eliminated by a long focal-screen distance. The paper tracing became a permanent record of cardiac size and contour. Obviously considerable individual variation was inherent in the method, but despite this drawback it had distinct advantages over the older non-roentgen methods and is still in use in some areas today.

At about this time, however, the danger to the roentgenologist of long fluoroscopic seances for study of the heart was recognized and by 1905 roentgenographic technique had advanced to such a degree that Köhler could introduce teleroentgenography for accurate measurement of cardiac size. Teleroentgenograms soon practically supplanted the time consuming and hazardous orthodiagraphic tracing method. Exposure time for roentgenography of the heart was still too long (*i.e.*, one-half second) as measured by present-day standards, and blurring of the cardiac borders on chest roentgenograms was accepted as a necessary evil but did not hinder further exploitation of roentgenography for cardiac diagnosis.

1906–1939

The first decade of this period saw reawakening of interest in the art of chest fluoroscopy. Perhaps stimulated by the French investigators who had already made solid progress in this direction, American radiologists returning from Europe began to more extensively apply fluoroscopy and subsequently roentgenography in oblique projections for the better demonstration of heart disease. Van Zwalenberg in 1919, voiced "a plea for the use of the fluoroscope in the examination of the heart and great vessels" in The American Journal of Roentgenology. Three years later the same thesis was editorialized under the title, *The Heart, the X-ray, and the Internist* by Augustus W. Crane, who noted that "it is not a generally accepted or much used method." He attributed this to the fact that internists are perhaps sufficiently supplied with instrumental means for examining the heart, many able and successful men holding that the palpating finger and the ear are the only instruments necessary. "The X-ray, to be sure, is accepted for the detection of aneurysm and to a less extent for the estimation of the size of the heart, but the same hard-headed conservatism which impeded the use of the thermometer, the stethoscope and other instrumental methods is still as active in some quarters as it was in the days of Wunderlich and Laënnec."

Doctor Crane quoted Sir James MacKenzie, world-renowned cardiologist, who throwing the weight of his reputation into a statement in one of the leading medical texts of that day (1920) opined that "in-

deed I am doubtful if an X-ray examination of the heart has ever thrown the slightest light upon any cardiac condition." Crane's reply for the roentgenologist is as alive today as when written a quarter of a century ago, when he said, "We do not undervaluate clinical methods, but such is the supremacy of the eye and such the importance of things seen that the roentgen-ray image will take an assured precedence as diagnostic evidence."

Despite the prejudices of some clinicians, gradually through the combined efforts of radiologists and cardiologists, the roentgen aspects of acquired heart disease were formulated. Holmes, for instance, elucidated the signs of pericardial disease; Pfahler and Baetjer classified intrathoracic aneurysms; Sosman detected and localized *in vivo* intracardiac calcifications; Zdansky, Roesler, Schwedel and many others correlated clinical, roentgenologic, and postmortem findings in valvular and other forms of acquired heart disease to lead to a better understanding of their roentgen manifestations. Congenital cardiac defects during this period were generally relegated to the storehouse of medical curiosities, with one glaring exception. In 1898 Zinn published an account of a case of *Persistence of the Ductus Arteriosus Botalli* in which the clinical diagnosis was confirmed by the fluoroscopic evidence of enlargement of the pulmonary artery. Thereafter, the enlarged pulmonary artery segment of patent ductus was called "the cap of Zinn."

In the 1920's roentgenologists became engrossed in cardiac mensuration. Le Wald and Bardeen had used simple transverse cardiac measurements compared to the transverse diameter of the chest at the cardiac level as an index of relative cardiac size. Eyster made similar observations and later both F. J. Hodges and P. C. Hodges and Eyster compared and related the transverse diameter of the heart to body height and weight and also related the frontal area of the heart to body height and weight. Bardeen had demonstrated a method for estimation of cardiac volume by roentgenography in 1924; Kahlstorf has evolved a formula for such volume determination which is used today particularly for assessing the results of therapy. More recently (1942) Ungerleider and Grubner prepared a nomogram of cardiac frontal area compared to body height and weight. Hilbish and Morgan (1952) have related frontal area of the heart to transverse diameter of the chest and Lind (1952) has correlated heart volume to body area in children.

An objective method for demonstrating the movements of the various portions of the heart and great vessels was first provided in 1911, several men contributing to the production of the roentgenkymograph —Sabat and Goett and Rosenthal are to be mentioned. However, Stumpf of Munich and I. S. Hirsch of the United States popularized kymography and contributed greatly to the better understanding of its complex curves. More recently, Boone and Henny, working in W. E. Chamberlain's laboratory, designed the electrokymograph which was later improved upon by Morgan and Sturm. The electrokymograph has proved more flexible than the roentgenkymograph and has been used quite extensively, both in this country and abroad, for the recording of cardiac dynamics.

In September, 1929, at the Thirtieth Annual Meeting of the American Roentgen Ray Society, a symposium on Cardiovascular Roentgenology was enjoined. The papers presented at this symposium indicate the level of interest at this period in that they all concerned anatomic features of acquired cardiac disease. The presentation was keynoted by Dr. Paul Dudley White who made "observations on the clinical value of the roentgen ray in the diagnosis of cardiovascular disease." Dr. White, then as now, one of the world's foremost cardiologists, listed seven such observations, excerpts from which follow: (1) Roentgenology is but one of several clinical methods of studying the circulation. (2) The roentgen rays provide a method for the demonstration of moderately or far advanced pathology only, even though

such pathology is revealed in no other way. (3) Serious and indeed fatal heart disease may be present with no indication of trouble on roentgen examination, or even by any other method of study except perhaps by history. This is particularly true of angina pectoris which is now generally recognized to be dependent primarily on coronary disease. (4) Roentgenology, even though at first glance giving the impression of being a very delicate and accurate method, results about equally in the occasional diagnosis of organic heart disease when none is present and in the failure to recognize such disease when it is present. (5) A method which is inherently accurate may become a source of confusion when errors creep in as they are so frequently likely to do in cardiovascular roentgenology, even in the best of hands. Here, Dr. White mentions improper positioning of the patient during roentgenography, distortion of the cardiac image with short focal-film distances and errors in measurement of the cardiac diameters. (6) The roentgenologist or internist using the roentgen rays should state what he sees in the way of structural change and not attempt to make an etiologic diagnosis. (7) The seventh observation concerns the actual method of examination, emphasis being laid on study in various positions.

Dr. White's appraisal in 1929 of the value of the roentgen ray in cardiology has been tempered somewhat in succeeding years, and in the latest volume of his famous text, *Heart Disease* (1951), he reiterates that cardiovascular roentgenology has become firmly entrenched as an important part of routine study of the heart and blood vessels, it ranking fourth in value as a method of examination after history taking, physical examination and electrocardiography.

Up until the early 1920's only the thoracic portion of the cardiovascular system could be satisfactorily studied roentgenologically. The need for some method of visualizing the peripheral portions of the vascular tree was quite evident and eventually solved when solutions containing various radiopaque media were introduced.

Contrast opacification of portions of the cardiovascular system began in 1923 with peripheral venography, and in June, 1924, Brooks of St. Louis demonstrated a femoral arteriogram made by the injection of sodium iodide directly into the artery. Egas Moniz in 1928 published the details of his method of carotid arteriography using a 25 per cent solution of sodium iodide. Moniz and other Portuguese investigators were soon able to demonstrate intracranial aneurysms and to localize brain tumors by this technique. Abdominal aortography was first practiced by dos Santos, Lamas, and Pereira Caldes of Lisbon in 1929. In the same year, Forssmann, a German physiologist, inserted an ureteral catheter into his own right atrium by way of an arm vein. Two years later he tried injecting an iodine compound (uroselectan) through a catheter lying in the right atrium and attempted to obtain roentgenographic visualization of the pulmonary vessels, but due to technical faults the films were unsatisfactory. Also in 1931, Moniz, Carvalho, and Lima used 120 per cent sodium iodide with the Forssmann technique and obtained satisfactory opacification of the pulmonary arterial system and occasionally the right heart chambers. This method they named angiopneumography.

Castellanos, Pereiras, and Garcia in 1937 devised a method whtich they called radiopaque angiocardiography. It consisted of intravenous injection of uroselectan B, followed by a single film exposure at the end of the injection. Their roentgenograms demonstrated the right heart very well, but the left heart was seen only in severe congenital cardiac anomalies where large shunts existed.

At the same time that these Cuban workers were making their early efforts, Robb and Steinberg were carrying out similar studies. They succeeded after they

had improved their technique by correlation of exposures with circulation times, the use of highly concentrated contrast medium, and the development of a wide-bore syringe and needle which permitted rapid injection of a large bolus of contrast medium into the selected vein. Opacification of the right and left heart chambers and the pulmonary vascular system could thus be regularly and rather simply obtained. Once these technical features were mastered Robb, Steinberg, and Dotter made extensive studies of the normal and abnormal angiocardiographic anatomy and physiology to establish a firm base for angiocardiographic interpretation. Selective angiocardiography and direct intracardiac injection of contrast medium are two recently described techniques which have not been extensively tried in this country.

Almost every portion of the vascular tree has now become susceptible to contrast visualization, giving rise to such methods as, for example, portal venography, splenoportography, transosseous venography, thoracic aortography, pelvic arteriography, caval phlebography, and dural sinus venography.

1939–1956

This period could logically be called the surgical era of cardiovascular roentgenology. Beginning with the successful ligation of a patent ductus arteriosus by Gross in 1939, rapid progress has been made in the surgical relief of congenital heart lesions. The radiologist has played no little part in this effort. It should be a source of satisfaction to the radiologist to recognize that much of the diagnostic information supplied to the surgeons is derived from roentgen studies. For example, Dr. Helen Taussig, the modern pioneer of congenital heart disease, leans heavily upon the roentgen ray, particularly the fluoroscope, for anatomic and physiologic information. Her combination of history, physical examination, and fluoroscopic study of the heart has allowed, in her hands at least, a remarkable degree of accuracy in the diagnosis of congenital heart disease. Her monumental volume, *Congenital Malformations of the Heart*, has served as a working manual for many roentgenologists and has stimulated further investigation of the roentgen features of the various congenital cardiac entities which she describes. Some radiologists have proceeded mainly along the lines of contrast studies of the heart (angiocardiography, aortography) while others believe, along with Sosman, that "the ultimate objective of all of these very complicated and sometimes dangerous procedures is to learn enough about these conditions so that we can go back and refer our knowledge to the original, simple, uncomplicated examination and know more about what we are seeing and finding." It has become evident that a combination of the simple and specialized roentgen techniques is necessary for a complete diagnosis in congenital heart disease. Coarctation of the aorta is a prime example of the evolution of such thinking.

The single most important roentgenologic sign of aortic coarctation, *i.e.*, rib notching, was described by Roesler in 1928. In 1930, Fray described a defect or break in the continuity of the aortic outline at the upper end of the descending limb of the arch. Wolke in 1937 and Bruwer and Pugh in 1952 confirmed that this indentation represents the coarctation site. Fleishner in 1949 described a displacement of the barium-filled esophagus to the right and anteriorly by the dilated poststenotic aortic segment. Angiocardiography has been employed in the diagnosis of aortic coarctation but frequently has failed to demonstrate the extent and exact anatomy of the coarcted segment. Thoracic aortography was used with great success by Jönsson and his co-workers in Sweden for this purpose. Sloan and Cooley, after reviewing their experience with a large series of coarctation of the aorta, concluded that contrast vascular techniques should be employed routinely only in those instances

where there is suspicion that the coarctation is atypical or where routine roentgenologic studies give no clear indication as to the site of the stenosis. Therefore, in their opinion, the preoperative roentgen diagnosis of aortic coarctation can be accomplished in most instances by the simple roentgen methods previously described.

Known to the anatomists and pathologists for many years, the major anomalies of the aortic arch and great vessels at the base of the heart had been relatively neglected roentgenologically until the possibility of surgical correction of these anomalies was suggested. Neuhauser in 1946 presented a practical classification of such anomalies and extensively discussed their roentgen diagnosis. In so doing he paid tribute to the earlier roentgenologists interested in this field, such as Kommerell, Renander, Arkin and Fray. The accurate diagnosis of the aortic arch anomalies was instrumental in permitting Gross in June, 1945 to be the first to successfully divide a vascular ring for relief of tracheal obstruction.

The rapid progress made in the surgical treatment of congenital malformations of the heart encouraged similar attempts at acquired lesions of the cardiovascular system. Roentgenology here again played a paramount role in ascertaining accurate preoperative diagnosis, establishing rigid criteria of operability and assaying precisely the improvement observed in the postoperative period as well as the long term curative results. Aneurysms, occlusive disease of the aorta and great vessels, and, more recently, lesions within the chambers of the heart itself are being attacked with increasing frequency of success, engendering a most enchanting and promising era of cardiovascular surgery.

Another major development of this era is the introduction of radioactive isotopes for the study of cardiovascular physiology and disease. Prinzmetal in 1949 described radiocardiography for detection of intracardiac shunts and other disturbances of intracardiac blood flow. By his technique radioactive sodium (Na^{24}) was injected intravenously and a continuous graphic recording of intracardiac radioactive blood concentration obtained by use of a shielded precordial Geiger-Mueller tube. The resulting diphasic curve represented a method for determining the pumping qualities of the heart, and has been particularly useful for detection of shunts. Circulation time in other parts of the cardiovascular system, arterial or venous, can also be studied by use of radioactive isotopes. Of special value has been the demonstration of slowed venous return in postsurgical patients by the use of radiosodium flow studies by Wright and Osborne of London.

Smith and Quimby, using Na^{24}, and Friedell and his co-workers, using P^{32}, plotted build-up concentrations following intravenous injection of these isotopes in tissues of extremities of patients with peripheral vascular disease and established an index thereby to judge the efficacy of therapy. Kety (1948) investigated the clearance of radioactive substances from muscle of an extremity and calculated a clearance constant for relation of blood flow to tissue clearance. This method, adopted by other investigators, has been found particularly useful as an index of circulation in pedicle skin grafts in plastic surgery and in the estimation of skin damage following radiation therapy (Roswit, Wisham and Sorrentino, 1953).

Blood volume, red cell mass and plasma volume can all be measured with the aid of radioactive isotopes. Radiochromium (Cr^{51}), radiophosphorus (P^{32}), and radioactive iron (Fe^{55} and Fe^{59}) have been used for this purpose. Radioactive sulfur (S^{35}), in the form of sulfate, is a satisfactory and convenient measure of the functional extracellular fluid volume.

Radioactive iodinated (I^{131}) human serum albumin is at present one of the agents employed for blood volume determination. Its use was initiated by Storaasli, Krieger, Friedell and Holden in 1950.

Many other isotopes and isotopic methods have been used and are presently being

investigated for application to the study of cardiovascular physiology and disease.

* * *

These past performances in cardiovascular radiology are impressive, and at present the roentgen method and all of its ramifications are firmly established and indispensable for the proper study of the cardiovascular system. Who can predict what the next fifty years will bring in cardiovascular radiology? Newer contrast media, better roentgenoscopic and roentgenographic techniques, more widespread use of radioactive substances, will undoubtedly play important parts, but most important of all will be the radiologist of the future who, taking his cues from his predecessors of the last half century, intelligently directs his diagnostic tools.

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PRACTICAL VALUE OF THE POSTEROANTERIOR ROENTGENOGRAM AND ROENTGENOSCOPY IN CERTAIN TYPES OF HEART DISEASE

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IT SHOULD be unnecessary to say that the roentgenologic interpretation of cardiovascular lesions can be satisfactorily done only on a basis of some knowledge of the more fundamental features of the anatomic defect and of the cardiodynamics.

Furthermore, any remarks about the *practical* value of the more routine roentgenologic methods in cardiologic conditions should be prefaced by recognition that the roentgenologist, using these methods, usually plays only a small, albeit definite, contributory role in the over-all picture of cardiologic diagnosis. It has been stated that he contributes about 10 per cent to the final diagnosis. It probably would be more accurate to say that the amount of help he provides varies from case to case. That is, in some conditions, such as the figure-of-eight type of anomalous pulmonary venous connection, the radiologist probably can make an entirely correct diagnosis by study of the posteroanterior thoracic roentgenogram alone, his contribution therefore being a large one. On the other hand, in arriving at a definitive diagnosis of the more complex types of congenital heart disease, such as tricuspid atresia with transposition or persistent common atrioventricular canal, his contribution will be much smaller.

What is important, if the method is to be useful, is for the roentgenologist to confine his statements within the limits that may serve some practical purpose in a particular case. It is the author's opinion that when roentgenograms are used to illustrate certain types of heart disease, they should be used merely as a step in the diagnosis, and that only the pertinent features should be emphasized. For example, if a roentgenogram is used to illustrate an instance of atrial septal defect, the accompanying legend should stress, not the fact that what is shown is a roentgenologic example of an atrial septal defect, but such features as the pulmonary trunk, the vascularity of the pulmonary fields and the appearance of the aortic shadow. These features may show a pattern commonly found in atrial septal defect. By correlation with other clinical and laboratory findings, the diagnosis is reached. To declare in a legend that a roentgenogram is that of, for instance, an atrial septal defect may be true, but is really somewhat deceptive. A similar roentgenologic picture may be seen in the presence of ventricular septal defect and persistent common atrioventricular canal, as well as other conditions. But the significant findings in the roentgenogram will be those which lead part of the way to the diagnosis. In other words, there will be very few times when the roentgenogram will be characteristic of a specific diagnosis in cases of congenital heart disease. But there will be many occasions on which the roentgenogram will furnish one or several building blocks in the completion of the final diagnostic structure.

Thus, for example, when the pulmonary fields show vascular engorgement of a certain type, several possibilities exist. If it is known that the patient is not cyanotic, the possibilities become fewer, and the existence of a left-to-right shunt becomes almost certain. If, in addition, it is seen that the aortic knob is large and that pulmonary vascular pulsations are not markedly accentuated during roentgenoscopy, the chances that a patent ductus arteriosus

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is present are excellent. If, on the other hand, the aortic knob is invisible and hilar and pulmonary pulsations are markedly increased, the chances are greatly in favor of a congenital intracardiac shunt, rather than a patent ductus arteriosus. Incidentally, much more reliance should be placed on these features than on the roentgenologic impression regarding ventricular size.

However, perplexity often arises when the question of a double lesion is considered. In such a contingency it behooves the roentgenologist to stay on firm ground, even if the patch of ground he stands on is small. As has been said, it is probably best to limit one's remarks to the significant features of the roentgenogram, and to suggest the conditions with which those features are consistent.

MATERIAL AND LIMITATIONS

A study of the thoracic roentgenograms of many hundreds of patients who came to the Mayo Clinic suffering from the more common types of acquired heart disease and certain types of congenital heart disease has been made. In all the cases considered, the condition was proved either at operation or at cardiac catheterization, or both. In study of these films it became apparent that much pertinent roentgenologic information can be obtained from a combined evaluation of the posteroanterior thoracic roentgenograms and roentgenoscopy of the hilar and peripheral pulmonary arteries. With few exceptions, other conditions in which roentgenoscopy may be of practical value are study of the size of the left atrium, detection of cardiac calcification, and especially valvular calcification, and in right aortic arch and anomalous right subclavian artery. Occasionally, a ventricular aneurysm can be demonstrated roentgenoscopically.

Ventricular enlargement in heart disease can be evaluated on the basis of posteroanterior thoracic roentgenograms, in many cases with considerable accuracy. On the other hand, evaluation of ventricular size by roentgenoscopic means often is so inaccurate, especially among patients with congenital heart disease, that attempts at roentgenoscopic evaluation are best not relied upon. It is also felt that roentgenoscopic interpretation based on an electrocardiographic report cannot be regarded as objective. Roentgenoscopic attempts at evaluation of individual ventricles in congenital heart disease often become separated from reality when it is realized that the heart under study may have essentially only one ventricle.

If evaluation of ventricular size were at all accurate, there would not, for instance, be the difficulty in distinguishing mitral stenosis from mitral regurgitation by roentgenologic means. The author has had the experience of making a fairly confident roentgenoscopic diagnosis of left ventricular enlargement in a case of mitral heart disease, only to learn that pure mitral stenosis was found at operation, and that the appearance of left ventricular enlargement apparently was due to moderate pericardial effusion.

Figure 1 shows a left anterior oblique view of an atrial septal defect in which a suggestion of left ventricular enlargement could be made easily and erroneously. Numerous such examples could be given.

The material presented herein has been restricted to those conditions in which it is felt that the roentgenologist can give either an unequivocal diagnosis, or to those conditions in which he can offer a helpful contribution to the final diagnosis. No attempt will be made to present any esoteric findings or to suggest findings that the examiner theoretically should be able to see, in view of a subsequent catheterization or necropsy. Therefore, no attempt has been made to interpret complex cardiac anomalies as they appear in roentgenograms.

Most of the roentgenograms that were analyzed pertained to adult persons and teen-age children. Yet some of the findings apply equally to roentgenograms of small children, although it is felt that accuracy of interpretation often is not as high when the patient is in the first few years of life as it

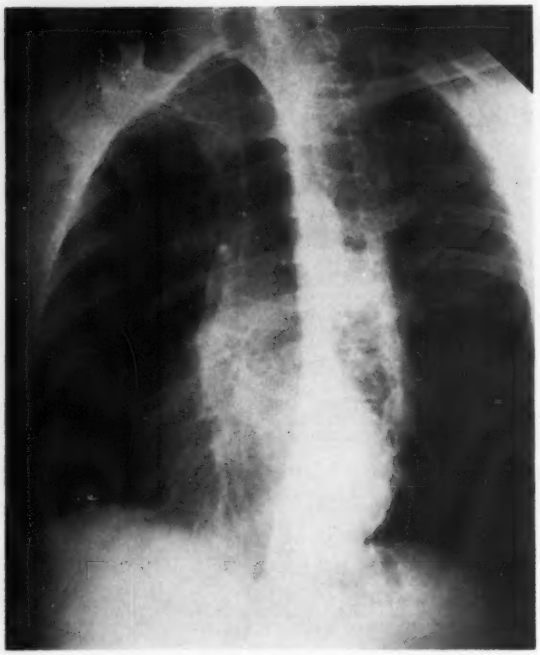

Fig. 1. Left anterior oblique thoracic roentgenogram of a patient who had atrial septal defect. This gives the erroneous impression of left ventricular enlargement. It is better to leave appraisal of the ventricles to the electrocardiographer than to rely on roentgenoscopy in such a case.

is when the patient is in the teen- or adult-age groups. As is well known among those who have dealt with roentgenograms of infants, it would not be surprising to find that, after a dozen roentgenograms of the thorax of one infant had been made in a single day, the heart has assumed different shape, and the lungs exhibit a different degree of aeration, in each roentgenogram. Furthermore, although the shadow cast by the thymus gland usually is easily recognizable, occasionally, as in the case illustrated in Figure 2, *a* and *b*, it can produce a roentgenologic picture that is most perplexing. In most cases of congenital heart disease in babies, it is best for the roentgenologist to evaluate the pulmonary fields for evidence of the degree of vascularity, such as diminished vascularity in the presence of obstruction of the pulmonary or tricuspid valve, or increased vascularity in the presence of septal defects and transposition. Evaluation of hilar pulsations or of ventricular size in infants is often impossible or is done with foreknowledge of ambiguous results. In an infant who has a large, failing

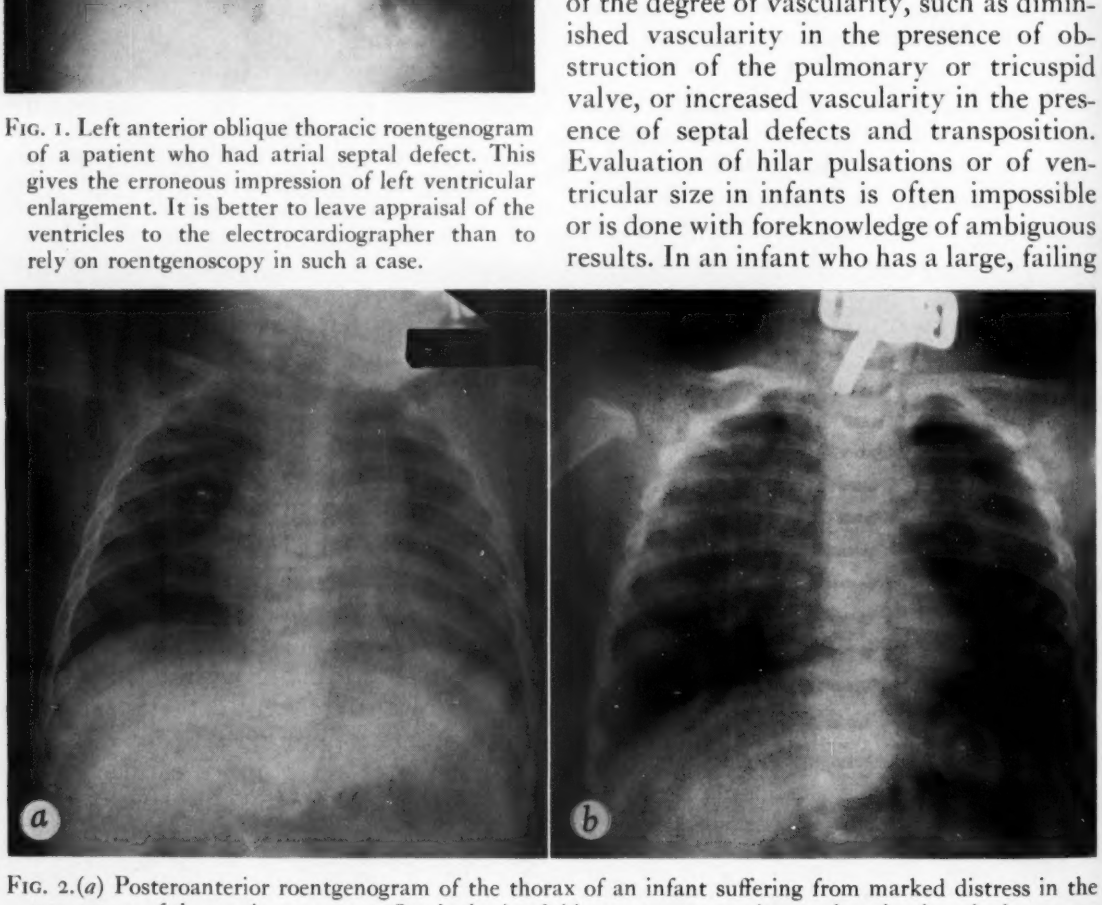

Fig. 2.(*a*) Posteroanterior roentgenogram of the thorax of an infant suffering from marked distress in the upper part of the respiratory tract. On the basis of this roentgenogram it was thought that the heart was enlarged. (*b*) Tracheotomy was done on the same day, and pneumothorax accidentally occurred. As a result of the pneumothorax, the lobes of the thymus gland became individually defined, and it is noted that what was previously thought to be cardiac enlargement was actually the result of an intimate blending of the shadows of the heart and thymus gland.

heart, a knowledge of whether or not the patient is cyanotic, together with the appearance of the pulmonary fields, will enable the examiner to categorize the lesion, leaving further investigation of the condition to the clinician and physiologist, perhaps with the help of more complicated roentgenologic procedures.

The features that have been regarded as valuable on the posteroanterior roentgenogram, other than among infants, include the following: First, the appearance of the shadow of the pulmonary trunk. (The term "pulmonary trunk" is used when reference is made to that part of the pulmonary artery projected near the left main bronchus.) Second, the appearance of that part of the aortic arch referred to as the "aortic knob." Third, the appearance of the branches of the pulmonary arteries as projected in the pulmonary fields. Fourth, the appearance of the border of the left side of the heart, according to whether it is more suggestive of right ventricular or left ventricular enlargement. In the former it tends to be "straight," and in the latter, "hollow." Fifth, the appearance of the border of the right side of the heart, with special reference to the double auricular contour produced in mitral heart disease. Sixth, the presence of costophrenic septal lines which are excellent and accurate indications of increased pulmonary venous pressure, whether the increase is caused by mitral heart disease or left ventricular failure. Seventh, the appearance of the proximal portion of the descending thoracic aorta. Finally, the finding of notching in the ribs.

Fig. 3. Posteroanterior roentgenogram illustrating "slight" enlargement of the pulmonary trunk (see text). This also shows "bulbous" aortic knob (see text).

GRADING OF PROMINENCE OF STRUCTURES

For descriptive purposes, the prominence of some of these features was graded. So that the reader may be apprised of the author's subjective interpretation, examples of the various grades are presented.

Pulmonary Trunk. This structure is referred to as being of *normal* appearance, or as being either *slightly*, *moderately*, or *markedly* enlarged. It is admitted that the dividing line between slight enlargement and normality is always subjective. Figure 3 shows *slight* enlargement of the pulmonary trunk. Figure 4 shows *moderate* enlargement of the pulmonary trunk. Figures 5 and 6 show *marked* enlargement of the pulmonary trunk.

Fig. 4. Posteroanterior roentgenogram illustrating "moderate" enlargement of the pulmonary trunk (see text). This figure also shows the "hypoplastic" aortic knob (see text).

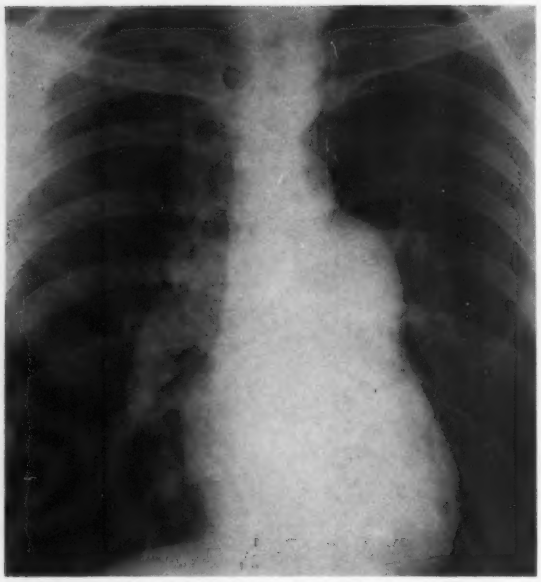

Fig. 5. Posteroanterior roentgenogram illustrating "marked" enlargement of the pulmonary trunk (see text). Marked pulmonary hypertension was present in this case.

Aortic Knob. The prominence of the aortic arch, seen to the left of the lower part of the trachea, is conveniently referred to as the "aortic knob." This structure is arbitrarily referred to as being *hypoplastic* when its shadow is absent or only very small on the posteroanterior roentgenogram. It should be stressed that roentgenograms of good penetration are required if the examiner is to be certain about the size of the aortic knob and the descending thoracic aorta. When the aortic knob is intermediate in size between the average, well-visualized *normal* knob and the hypoplastic type, it is referred to as *small.* These terms are not meant to indicate that the aortic knob actually is hypoplastic or small anatomically; they merely describe the comparative appearance of the shadow of the aortic knob in the posteroanterior roentgenogram. The term *bulbous* has been used to describe the particular type of enlargement of the aortic knob frequently seen in the presence of patent ductus arteriosus. Figure 7, *a* and *b*, shows a *small* aortic knob. Figure 8, *a* and *b*, shows a *normal* aortic knob. Figure 4 depicts a *hypoplastic* aortic knob. Figures 3 and 9, *a* and *b* demonstrate a *bulbous* aortic knob.

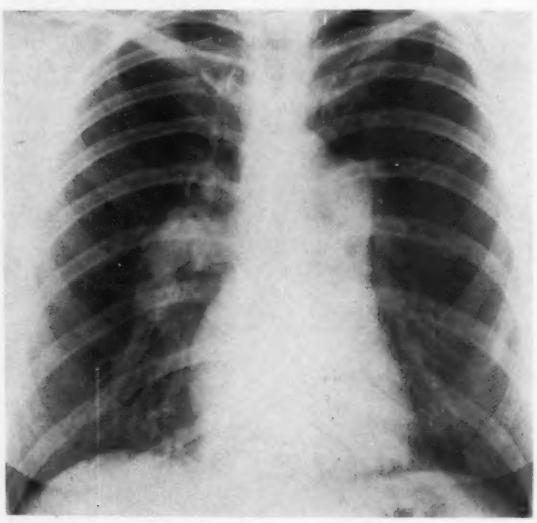

Fig. 6. Posteroanterior roentgenogram illustrating "marked" enlargement of the pulmonary trunk (see text). This is a case of idiopathic pulmonary hypertension in which pulmonary venous hypertension is not present.

Vascularity of the Pulmonary Fields. It is well known that the appearance of the vascular supply of the pulmonary fields differs considerably in various cardiac conditions. Thus, in pulmonic stenosis the posteroanterior roentgenogram may reflect the diminished flow of blood through the lungs, whereas in the presence of a left-to-right shunt, such as in patent ductus arteriosus or in atrial septal defect, there may be evidence of the increased amount of blood flowing through the lungs. The latter appearance sometimes is referred to as "pulmonary congestion." By no means do all congenital cardiac conditions associated with a left-to-right shunt exhibit roentgenologic evidence of the shunt in the pulmonary fields. If the terms "congestion" or "congested pulmonary fields" are to be used, it should be stressed that the roentgenologic picture of congestion, as seen in conditions in which a shunt is present, usually is considerably different from the roentgenologic picture of pulmonary congestion seen, for instance, in the presence of mitral heart disease or cardiac failure. When such evidence is present in congeni-

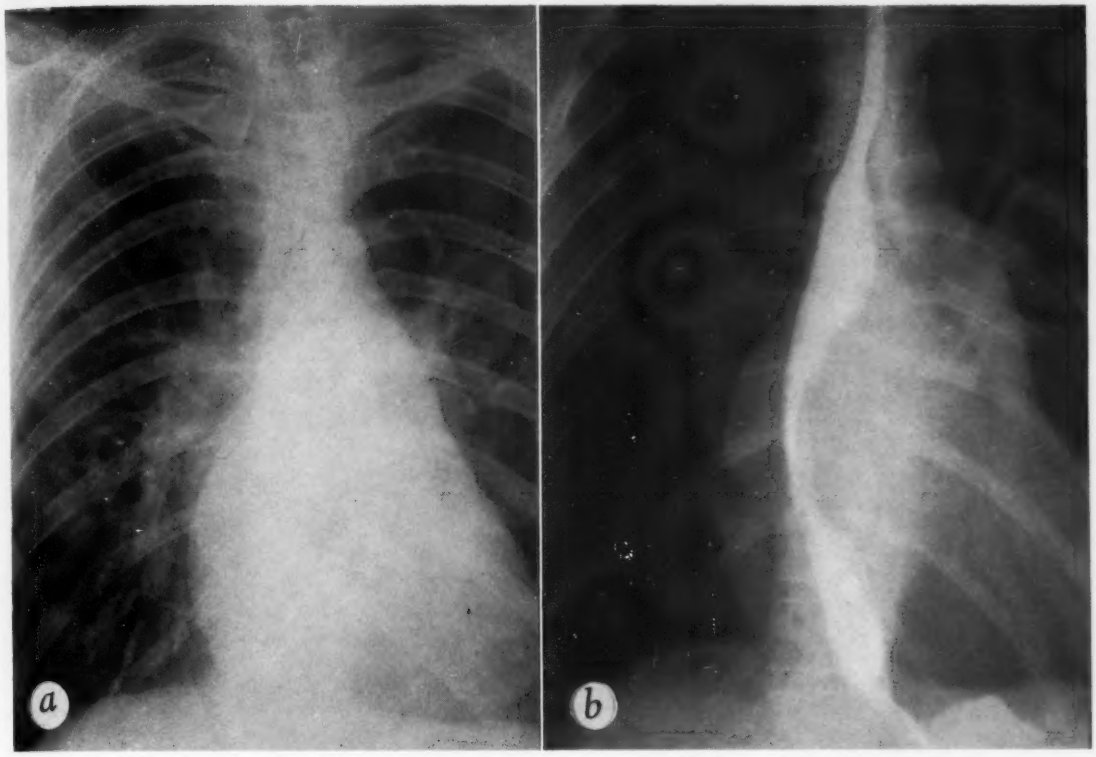

Fig. 7.(*a* and *b*) Posteroanterior roentgenogram and supine roentgenogram of patient who had mitral stenosis, depicting a "small" aortic knob.

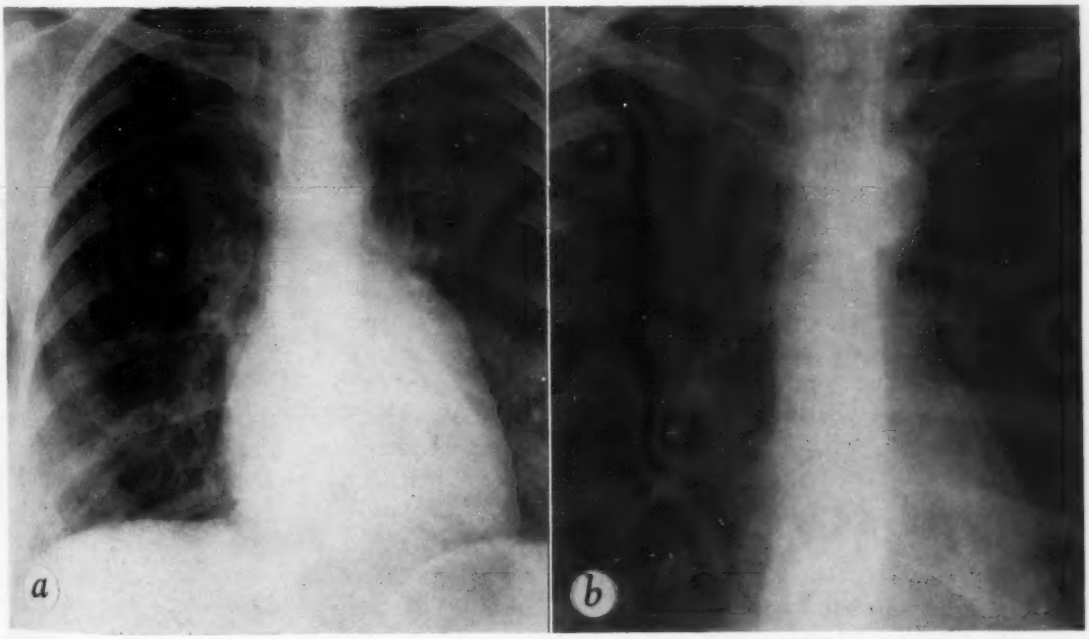

Fig. 8.(*a* and *b*) Posteroanterior thoracic roentgenograms illustrating "normal" aortic knob (see text).

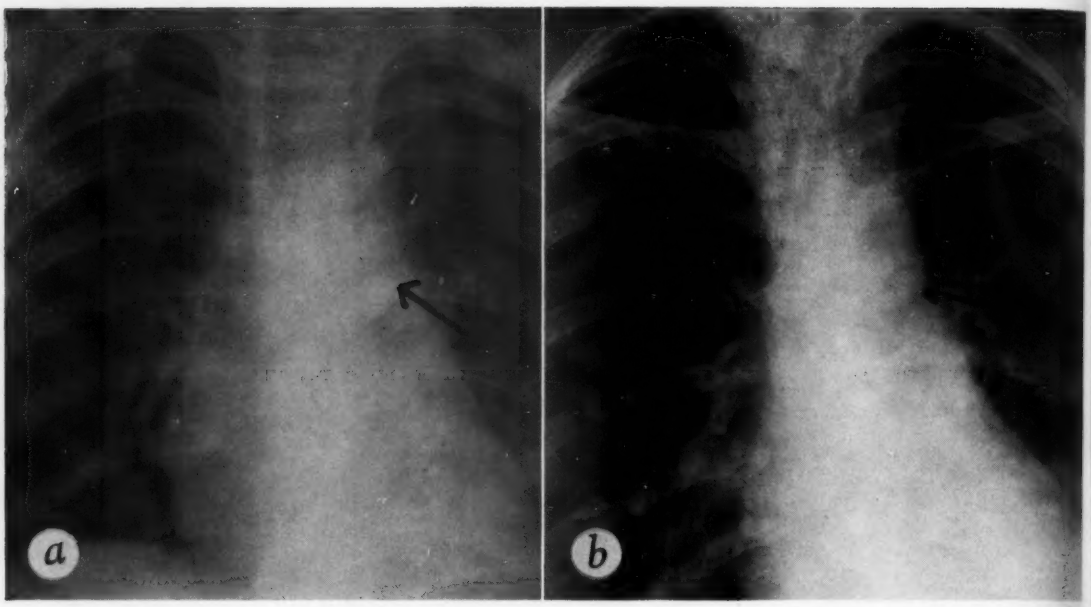

Fig. 9. (*a* and *b*) Posteroanterior roentgenograms illustrating "bulbous" aortic knob (see text).

tal heart disease, the pulmonary arteries and their branches have a fairly easily recognizable appearance. The pulmonary trunk usually is enlarged, and the appearance of the first branches of the pulmonary arteries suggests vessels engorged with blood. Generally, these vessels are *sharply defined,* and their various engorged branches often can be traced to the peripheral pulmonary fields, being larger and plumper than usual in their entire extent. Although the main pulmonary branches in the hilar regions may be markedly enlarged, as a rule they are rather well defined. This appearance is often more striking when pulmonary hypertension and pulmonary arteriolar obliterative changes are superimposed on conditions in which a congenital left-to-right shunt is present, and the contrast becomes evident when roentgenograms of a person with such a condition are compared with those of a patient who has mitral stenosis with pulmonary hypertension. In the latter condition the appearance of the vascular structures is modified, probably because of a diffuse increase in the hemosiderin and fibrous-tissue content of the lung, sometimes giving rise to a diffuse, frosted-glass appearance. Furthermore, in the presence of mitral heart disease the pulmonary veins, as well as the pulmonary arteries, are overfilled with blood at a pressure which is considerably higher than normal, and the lymphatic vessels also are engorged. This engorged condition of the pulmonary arteries, veins and lymphatic vessels may give rise to an image in which the right hilus may even resemble an irregular mass. In conditions associated with an intracardiac left-to-right shunt, the right hilar arteries also may be very prominent. But when pulmonary hypertension exists in the latter group, the nature of the process that aggravates the hypertension (that is, the obliterative changes in the small peripheral pulmonary arterioles) gives rise to a situation in which the pulmonary veins are spared, so that they do not share the hypertension as they do in mitral heart disease. The arterial without venous distribution of the pulmonary hypertension in the intracardiac left-to-right shunt condition also probably does not result in engorged lymphatics such as occurs in mitral heart disease. The difference between pulmonary arterial hypertension, as opposed to pulmonary arterial hypertension plus venous hypertension plus engorged lymphatic vessels, is well reflected in the posteroanterior thoracic roentgenogram in the

right paracardiac region. In the former condition this area tends to be clear; in the latter condition (mitral heart disease) this area tends to be hazy. This is probably because, when the latter condition is present, this area is occupied by markedly distended pulmonary veins passing medially to the left atrium as well as being densified by the over-all haziness of the pulmonary fields often found in mitral heart disease (hemosiderosis and lymphatic congestion). These differences are illustrated in Figures 5, 6 and 10, *a*, *b*, and *c*.

The appearance of the vascularity of the pulmonary fields in the posteroanterior thoracic roentgenogram has been graded as follows.

1. Reduced. The vascularity of the pulmonary fields was graded as "reduced" only when it was felt that the finding was unequivocal. Perhaps the criterion was applied overstrictly, this being the reason why such a small percentage of roentgenograms of our patients with pulmonic stenosis had a reduced pulmonary vascularity. Figure 11 illustrates unequivocal reduction of pulmonary blood flow in a case of tricuspid atresia.

2. Normal. A fairly wide range in the appearance of pulmonary vascularity was

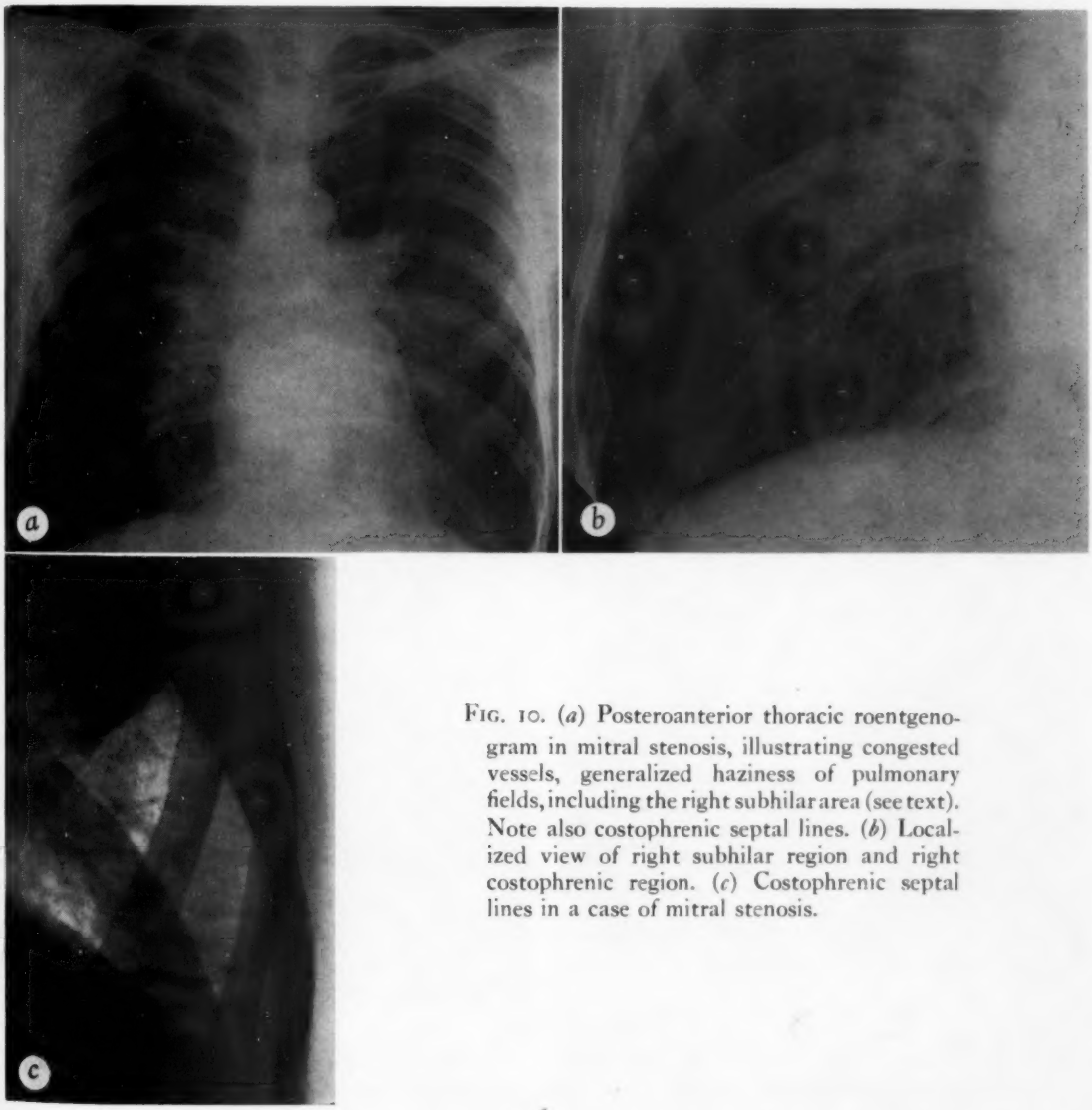

FIG. 10. (*a*) Posteroanterior thoracic roentgenogram in mitral stenosis, illustrating congested vessels, generalized haziness of pulmonary fields, including the right subhilar area (see text). Note also costophrenic septal lines. (*b*) Localized view of right subhilar region and right costophrenic region. (*c*) Costophrenic septal lines in a case of mitral stenosis.

FIG. 11. Posteroanterior roentgenogram of the thorax of a child, showing unequivocal "reduced" vascularity of the pulmonary fields (see text). The condition was tricuspid atresia.

classified as "normal," it being felt that to be of definite roentgenologic value, any change from the normal should be obvious and not unduly equivocal.

3. Increased. Increased vascularity of the pulmonary fields was graded as *slight*, *moderate* and *marked*. Examples of some of the gradations are illustrated. *Slightly increased* vascularity is shown in Figure 12; *moderately increased* vascularity is shown in Figure 13; and *markedly increased* vascularity is evident in Figure 14.

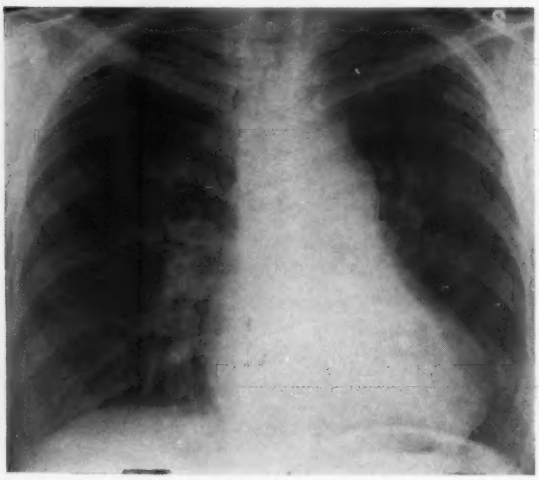

FIG. 12. Posteroanterior roentgenogram of the thorax, illustrating "slight" increase in the pulmonary vascularity (see text). This was the result of an intracardiac left-to-right shunt.

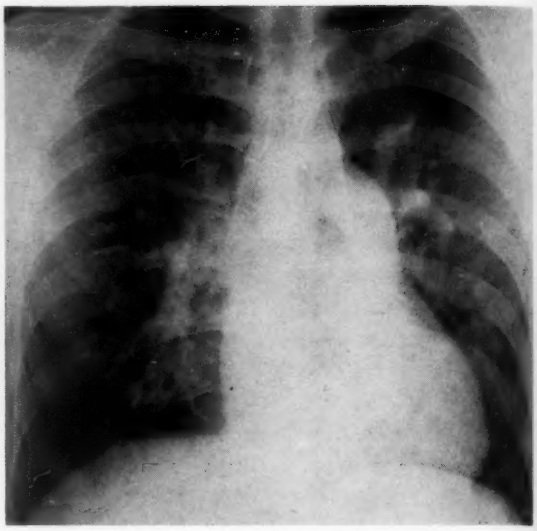

FIG. 13. Posteroanterior roentgenogram of the thorax, showing "moderately" increased pulmonary vascularity (see text). This appearance was the result of an intracardiac left-to-right shunt.

ROENTGENOSCOPY

As has been stated previously, roentgenoscopic study in congenital heart disease is thought to contribute most by enabling evaluation of pulsations of the hilar and more peripheral pulmonary arteries. At the

FIG. 14. Posteroanterior roentgenogram illustrating "marked" increase in the pulmonary vascularity. The condition was atrial septal defect.

Mayo Clinic several examiners perform cardiac roentgenoscopy. It has, therefore, been necessary to evaluate the degree of pulmonary vascular pulsation in these cases by interpreting the descriptions of several examiners. Admittedly, this permits error, and so it was thought best to limit the grading of pulsation to broader groups. These groups are as follows: (1) *normal* degree of pulsation; (2) *moderate* increase in hilar and peripheral pulmonary arterial pulsation; and (3) *marked* increase of hilar and peripheral pulmonary arterial pulsations or "hilar dance."

The demonstration of enlargement of the *left atrium* is an extremely reliable roentgenoscopic finding, even in infants. Left atrial enlargement as a rule is easily demonstrable by the patient's swallowing of barium, unless a grossly enlarged heart is present. The determination of *valvular calcification* also is considered to be a very accurate roentgenoscopic finding (provided the examiner is thoroughly adapted to the dark), and it is felt that the identity of the valve can be established with considerable accuracy by the technique described by Sosman, if either mitral or aortic valvular disease is predominant. Generally, myocardial and pericardial calcification is easily determined. As has been said previously, roentgenoscopic evaluation of ventricular size very often is faulty, so that it is probably best relegated to a minor role in cardiac roentgenology. In acquired heart disease roentgenoscopy of the ventricles should be relied upon only if it produces unequivocal results, whereas the method ought not to be relied upon in congenital heart disease. Sufficient and more nearly accurate information can be obtained from the posteroanterior roentgenogram, with occasional reference to oblique and lateral roentgenograms. Furthermore, electrocardiography is a more accurate method of estimating ventricular size than is either roentgenoscopy or roentgenography.

CONDITIONS REPRESENTED IN THIS STUDY

Congenital Cardiovascular Conditions. 1. Atrial Septal Defect. The roentgenograms of 61 patients who had atrial septal defect, without any associated cardiac lesion, were examined. In all these the condition had been proved at cardiac catheterization or operation or both. The features noted in the thoracic roentgenogram were as follows. In 29 cases the cardiothoracic ratio was 50 per cent or less, and in only 6 cases was there a cardiothoracic ratio of more than 60 per cent. The size of the heart in itself is of no practical value in arrival at a diagnosis of atrial septal defect.

Size of the Pulmonary Trunk. In 8 per cent of cases the pulmonary trunk was normal in appearance; in 19 per cent it was slightly enlarged; in 52 per cent of cases it was thought to be moderately enlarged, and in 21 per cent of cases the enlargement was considered marked. It is seen, therefore, that there was considerable enlargement of the pulmonary trunk in 73 per cent of the cases.

Aortic Knob. The aortic knob was considered to be of normal appearance in 26 per cent of cases. In 74 per cent of cases it was considered to be smaller than normal, the grading being "small" in 30 per cent of cases and "hypoplastic" in 44 per cent of cases.

Pulmonary Vascular Markings. In 25 per cent of cases pulmonary vascular markings appeared normal, in 35 per cent the vascularity was *slightly* but definitely increased, in 27 per cent of cases the increase was *moderate* and in 13 per cent it was graded as *marked.*

Pulmonary Arterial Pulsation (45 Cases). Pulmonary arterial pulsation did not evoke any unusual comment in 18 per cent of cases. In 11 per cent of cases pulsations were slightly increased. In 55 per cent of cases they were moderately increased, while in 16 per cent of cases pulsations were markedly increased.

Briefly, then, in atrial septal defect, the pulmonary trunk was enlarged in 92 per cent of cases, the aorta was smaller than normal in 74 per cent of cases, the pulmonary fields showed evidence of an increased content of blood in 75 per cent of cases, and the roentgenoscope showed significant in-

crease in the pulsations of the hilar and intrapulmonary arteries in 71 per cent of cases.

2. Ventricular Septal Defect. The roentgenograms of 26 patients in whom ventricular septal defect was the only cardiac lesion were studied. In only 6 of these cases was the cardiothoracic ratio more than 60 per cent, while in 11 cases it was 50 per cent or less. The size of the heart is of no practical value in the diagnosis of ventricular septal defect.

Size of the Pulmonary Trunk. In 33 per cent of cases the pulmonary trunk was normal in appearance; in 8 per cent of cases it was slightly enlarged, in 38 per cent of cases the enlargement was moderate, while in 21 per cent of cases it was markedly enlarged.

Aortic Knob. The aortic knob was considered to be normal in 27 per cent of cases. It was smaller than normal in 73 per cent of cases, the grading being "small" in 27 per cent of cases and "hypoplastic" in 46 per cent of cases.

Pulmonary Vascular Markings. In 27 per cent of cases the pulmonary vascular markings were normal, in 19 per cent of cases they were slightly increased, in 46 per cent of cases they were moderately increased, while in 8 per cent of cases they were markedly increased.

Pulmonary Arterial Pulsations. In 20 of the cases roentgenoscopy was done. Pulmonary arterial pulsations were not regarded as abnormal in 25 per cent of cases. In 3 per cent of cases the pulsations were interpreted as being slightly increased, in 50 per cent of cases the increase was moderate, and in 10 per cent the pulsations were marked.

In summary then, in ventricular septal defect the pulmonary trunk was enlarged in 67 per cent of cases (as opposed to 92 per cent in atrial septal defect); the aorta appeared to be smaller than normal in 73 per cent of cases (74 per cent in atrial septal defect); the pulmonary fields showed evidence of increased blood flow in 73 per cent of cases (75 per cent in atrial septal defect); and the roentgenoscope showed a significant increase in pulsations in the hilar and intrapulmonary arteries in 60 per cent of cases (71 per cent in atrial septal defect).

3. Patent Ductus Arteriosus. This is the third major, primarily acyanotic, condition in which a shunting of blood occurs through the pulmonary fields. The roentgenograms of 99 patients with patent ductus arteriosus proved at operation and in whom no other cardiac abnormality was present were studied.

Again, the cardiothoracic ratio was used as a rough measure of cardiac size. In 41 cases the ratio was 50 per cent or less, in 39 cases it was between 50 per cent and 60 per cent, while in 19 cases it was more than 60 per cent. In this group of cases of patent ductus arteriosus, there were 30 with moderate-to-marked pulmonary hypertension.

It is of interest that among these 30 cases, the cardiothoracic ratio was more than 60 per cent in 15 cases, while it was 50 per cent or more in 27 of the 30 cases. In all 30 cases pulmonary hypertension had been proved by cardiac catheterization. Among the remaining 69 cases were several in which the surgical description was characteristic of pulmonary hypertension, and it was noted that in these cases the heart also tended to be large. It is therefore interesting to find that of the group of three conditions usually characterized by a left-to-right shunt (atrial septal defect, ventricular septal defect and patent ductus arteriosus), the group in which marked cardiac enlargement occurs is that of patent ductus arteriosus. Furthermore, the cases of patent ductus arteriosus in which marked cardiac enlargement is seen also are the cases in which significant pulmonary hypertension is present.

Size of the Pulmonary Trunk. In 43 per cent of cases the size of the pulmonary trunk was thought to be within normal limits, whereas in 24 per cent it was considered to be *slightly* enlarged, in 29 per cent *moderately* enlarged, and in 4 per cent *markedly* enlarged. Definite enlargement of the pulmonary trunk was therefore thought to be present in 57 per cent of cases.

Aortic Knob. In only 7 per cent of cases

of patent ductus arteriosus was the aortic knob graded as *hypoplastic*, and in 8 per cent it was graded *small*. In 52 per cent of cases it was graded as of *normal* prominence, while in 33 per cent it was considered to appear *bulbous*. This appearance of an unusually massive aortic knob no doubt is related to the presence of the infundibulum of the ductus as described by Jönsson.

Pulmonary Vascular Markings. Pulmonary vascular markings were thought to be within normal limits in 60 per cent of the cases. In 40 per cent there was some increase in the vascular markings, this group being made up as follows: in 14 per cent, a *slight* increase in the markings; in 19 per cent a *moderate* increase in markings; and in 7 per cent a *marked* increase in the markings. It was of further interest that when pulmonary hypertension was present, moderate-to-marked evidence of re-circulation was seen in the pulmonary fields, whereas in only 21 per cent of cases in which there was no pulmonary hypertension was there moderate-to-marked evidence of re-circulation in the pulmonary fields.

Pulmonary Arterial Pulsations. The roentgenoscope was used in 56 cases of patent ductus arteriosus. In 68 per cent of these cases there was no evidence of an unusual degree of pulsation in the pulmonary trunk or its branches. In 12 per cent there was evidence of *slight* increase in the amplitude of pulsations; in 18 per cent the increase was *moderate;* while in 2 per cent it was thought to be *markedly* increased. It was therefore only in a fifth of cases of patent ductus arteriosus that roentgenoscopy revealed evidence of marked-to-moderate increase in pulmonary arterial pulsations.

Left atrial enlargement was noted in only 3 of the cases in which roentgenoscopy was used.

Comment on Atrial Septal Defect, Ventricular Septal Defect and Patent Ductus Arteriosus. It can be noted from the findings in the three foregoing conditions that there exist too many similarities in the roentgenologic findings of atrial septal defect and ventricular septal defect for the radiologist to be able to claim an ability to distinguish these conditions by means of routine roentgenologic methods.

It is noted, however, that patent ductus arteriosus tends to produce a different pattern. Among the three conditions in question, the largest hearts are found in patent ductus arteriosus, and it would seem that with a cardiothoracic ratio of more than 50 per cent in patent ductus arteriosus, moderate-to-severe pulmonary hypertension is likely to be present. Furthermore, study of the aortic knob, of pulmonary vascularity and of pulmonary arterial pulsations shows significant differences as these features are seen roentgenologically in intracardiac septal defect as opposed to patent ductus arteriosus (Table 1).

Thus, the aortic knob was small or hypoplastic in only 15 per cent of cases of patent ductus arteriosus, and actually was

Table I

DIFFERING ROENTGENOLOGIC OBSERVATIONS AS MADE IN INTRACARDIAC SEPTAL DEFECT COMPARED WITH PATENT DUCTUS ARTERIOSUS

Diagnostic Feature	Percentile Occurrence in		
	Atrial Septal Defect	Ventricular Septal Defect	Patent Ductus Arteriosus
Pulmonary trunk enlarged	92	67	57
Aortic knob smaller than normal	74	73	15*
Increased vascularity of pulmonary fields	75	73	40
Pulmonary arterial pulsations increased moderately to markedly	71	60	20

* Not only was the aortic knob usually not small, but in 33 per cent of cases it was bulbous.

bulbous in a third of cases. The vascularity of the pulmonary fields as a rule is not particularly increased in patent ductus, whereas in intracardiac septal defects it generally is increased. As to the roentgenoscopic appearance of the pulmonary arterial tree, it is noted that pulsations in intracardiac conditions in which a shunt is present usually are increased moderately or markedly, whereas in patent ductus arteriosus the increase was moderate or marked in only 20 per cent of cases. It has been interesting to find that, for the same amount of blood shunted from left to right, a patient with atrial septal defect will show considerably more pulmonary arterial pulsation than will a patient who has patent ductus arteriosus. The reason for this probably is the fact that, given an equal left-to-right shunt, in the presence of patent ductus arteriosus the shunt takes place from the aorta to the pulmonary arterial system throughout systole and diastole, whereas in atrial septal defect (and ventricular septal defect) the amount of re-circulating blood is shunted into the pulmonary artery during systole only. It would therefore be expected that more roentgenoscopic activity of the pulmonary arterial tree would be noted in intracardiac septal defects than in patent ductus arteriosus.

It is known that the conditions which commence with a lesion which results in a left-to-right shunt are capable of developing pulmonary hypertension as a complication. It has also been shown that pulmonary hypertension is often associated with obliterative changes in the finer branches of the peripheral pulmonary arteries. That the obliteration of a vast portion of the peripheral pulmonary arterial bed may be reflected in the roentgenogram of the thorax is suggested by the appearance frequently seen in pulmonary hypertension, namely, an apparent marked diminution of peripheral pulmonary vascularity in the face of large, congested pulmonary hilar arteries. This point is illustrated by a comparison of the peripheral pulmonary fields in Figure 5 with those in Figure 14. In both instances the condition is atrial septal defect; and in the former marked pulmonary hypertension was present, while in the latter (Fig. 14) pressures in the pulmonary artery were normal. It should be noted that there is an important difference between Figures 5 and 15, *a* and *b;* in both the peripheral pulmonary fields are relatively clear, while the pulmonary trunk is large. Speculation might be made as to the presence of pulmonary hypertension in both cases; the important point is that in Figure 15, *a* and *b*, which illustrates pulmonary valvular stenosis with poststenotic dilatation, the right hilar arteries are very small compared to the left. It is not intended to imply that the examiner can always determine whether pulmonary hypertension has supervened in a condition that may have started off as a purely left-to-right shunt. However, the features discussed make it possible to present the suggestion with reasonable certainty.

4. Pulmonic Stenosis With Intact Ventricular Septum. The roentgenograms of 33 patients were examined. In all cases the condition had been proved either by cardiac catheterization or at operation or both. In 8 the stenosis was valvular only, in 4 it was infundibular only, and in 3 a combination of types existed. In 17 cases the type was unknown. In 10 of the 33 cases an atrial septal defect or valve-incompetent foramen ovale was known to exist, but these factors do not affect the roentgenologic appearance significantly in these cases.

The heart usually is not enlarged, as judged by the cardiothoracic ratio. In 26 of the 33 cases the cardiothoracic ratio was 50 per cent or less.

Pulmonary Trunk. In 18 per cent of cases the pulmonary trunk was of normal appearance, and in another 18 per cent it was slightly enlarged. Moderate enlargement was noted in 55 per cent of cases, and marked enlargement in 9 per cent. It is to be noted that when the pulmonary trunk and the first portion of the left pulmonary artery appear large, there is no corresponding enlargement of the pulmonary artery in

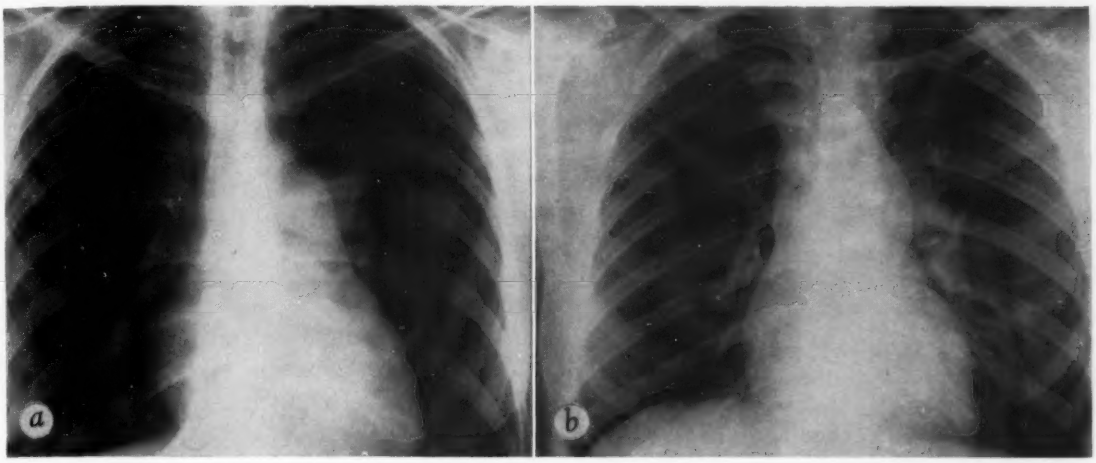

Fig. 15.(*a*) Posteroanterior roentgenogram of the thorax, illustrating moderate enlargement of the pulmonary trunk, as well as of the left main pulmonary artery. Note, however, that the right pulmonary artery is not correspondingly enlarged and is in fact small in contrast to the pulmonary trunk and left pulmonary artery. These features are therefore extremely suggestive of pulmonic stenosis with post-stenotic dilatation on the left. The anemic appearance of the pulmonary fields is another striking feature in this case. (b) A similar case.

the right hilus. In fact, the pulmonary artery in the right hilus may appear to be unduly small compared to the appearance of the artery in the left hilus (Fig. 15, *a* and *b*).

Aortic Knob. The aortic knob was found to be hypoplastic in 29 per cent of cases; it was small in 21 per cent of cases and normal in 50 per cent of cases.

Pulmonary Vascular Markings. In 90 per cent of cases the pulmonary vascular markings were graded as normal. It is possible that this percentage is much too high, but it was thought that unless the finding of decreased pulmonary vascularity was unequivocal, it was preferable to grade the vascularity as normal. In only about 7 per cent of cases was the pulmonary vascularity thought to be definitely diminished. In 3 per cent of cases the vascularity of the pulmonary fields gave the appearance of being slightly increased.

Pulmonary Arterial Pulsations. The pulsations of the pulmonary arteries in the hilar regions were graded as within normal limits in 92 per cent of cases. In the other 8 per cent a note was made that the pulmonary arterial structures in the left hilus pulsated considerably more than those in the right hilar regions. This is a finding which Blount has reported in all of his cases of pulmonic stenosis. Glover also has mentioned it. Both these workers suggest that the increase in pulsations of pulmonary arterial structures in the left hilus is due to the passing of the jet of blood through the stenosed pulmonic valve or subpulmonary region; the fast, thin stream of blood causes a visible movement of the pulmonary trunk in the left hilus. We have noticed this finding, and with greater consciousness of it probably will record it more frequently. This increase of pulmonary arterial pulsations on the left, however, can be mimicked by other conditions, and the author has seen the same appearance in a case of Ebstein's malformation of the tricuspid valve associated with an atrial septal defect.

Comment on Pulmonic Stenosis with Intact Ventricular Septum. Criteria that are of practical value in evaluating a case from the point of view of pulmonic stenosis with intact ventricular septum are: the frequent occurrence of a moderate degree of enlargement of the pulmonary trunk, and sometimes of the first segment of the left pulmonary artery; in comparison with the

prominence of the appearance of the pulmonary arterial structures in the left hilar region, the pulmonary artery in the right hilar region often appears normal or even small. The peripheral pulmonary arterial supply may be considerably diminished, but if the roentgenologic finding is to be useful and accurate, this diminution should be definite and not equivocal. The posteroanterior appearance of the heart may, by the prominence of the border of the left side of the heart and absence of the cardiac waist, give ample evidence of right ventricular enlargement. If there is doubt, a lateral view may be helpful to show encroachment of the cardiac substance in the retrosternal space. Roentgenoscopy of the ventricles in the oblique positions will add nothing or very little to what can be adjudged from the posteroanterior and lateral thoracic roentgenograms; moreover, roentgenoscopy may lead to marked errors in the interpretation of ventricular size.

5. Tetralogy of Fallot. The cardiac configuration on the posteroanterior roentgenogram in tetralogy of Fallot is so variable that little reliance can be placed on such catch phrases as *cœur-en-sabot* to lead to a correct diagnosis. The elevated cardiac apex of the *cœur-en-sabot* may be seen in both tricuspid atresia and in tetralogy of Fallot, and it is, of course, a different ventricle which is predominant in the two conditions.

The cardiothoracic ratio suggests no striking pattern of cardiac enlargement. In fact, the cardiothoracic ratio usually is close to 50 per cent.

Pulmonary Trunk. In 25 per cent of cases the shadow of the pulmonary trunk was thought to be smaller than normal. In 66 per cent of cases it appeared to be within normal limits of size, while in 6 per cent and 3 per cent of cases, respectively, it appeared to be slightly and moderately larger than normal, probably as a result of some poststenotic dilatation.

Aorta. In 78 per cent of 23 cases in which the aortic knob could be distinguished on posteroanterior thoracic roentgenograms, the knob was graded as *normal*. In 14 per cent it was graded as *bulbous*, while in 8 per cent it was thought to appear *small*. In 6 cases the size of the knob could not be judged because of unsatisfactory penetration. Surgical and roentgenologic findings in 28 cases revealed the aortic arch on the right side in 10 cases.

Appearance of Pulmonary Fields. In 75 per cent the appearance of the pulmonary fields was graded as within normal limits. In 19 per cent of cases there was evidence of decreased pulmonary markings, while in one case each the markings were thought to be slightly and moderately increased. Although there was a suggestion that the blood supply of the lungs had a somewhat unusual and more erratic course than the usual regular branching of the pulmonary arteries, this appearance, suggesting large bronchial arteries, was striking in only a few cases.

Roentgenoscopic Appearance. In the cases in which roentgenoscopy was used, the hilar pulsations were thought to be within normal limits. It was of interest that in the majority of cases in which roentgenoscopy was employed, the left ventricle alone was said to be enlarged. This demonstrates again the futility of attempts to judge ventricular size by roentgenoscopy, especially in congenital heart disease.

6. Coarctation of the Aorta. The two most common roentgenologic features of coarctation of the aorta generally are satisfactorily seen in the routine posteroanterior roentgenogram (Fig. 16, *a* and *b*, 17, *a*, *b* and *c*). As a rule, notching of the vascular grooves of the ribs by the tortuous intercostal arteries is easy to recognize. However, another sign that can be seen in the roentgenogram in a third of cases of coarctation is the actual site of the coarctation. It appears as a notch in the contour of the upper part of the descending aorta, and we noticed it in about a third of cases of coarctation. In 5 per cent of cases of coarctation this sign was present when notching of the ribs was not evident. It is especially valuable in the presence of coarctation among

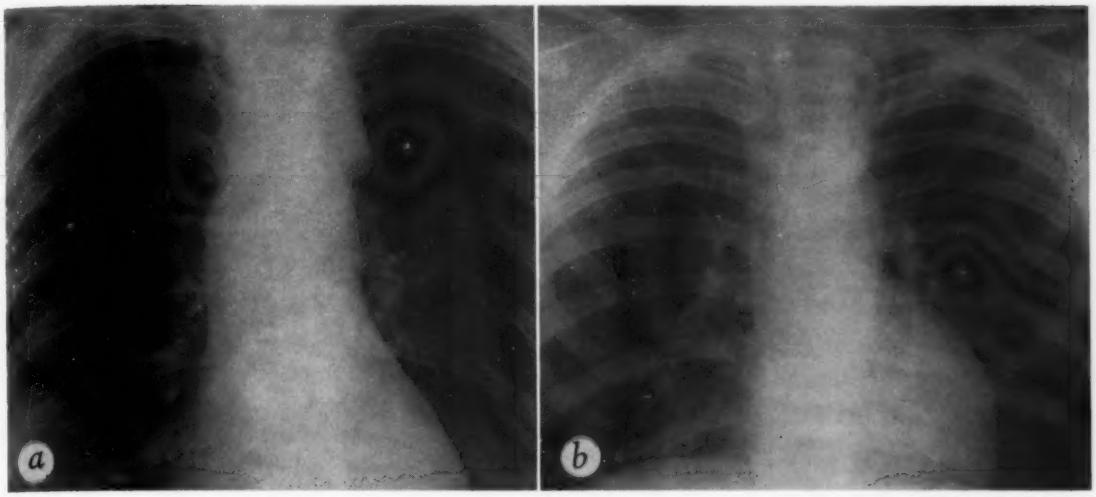

Fig. 16. (*a*) Posteroanterior roentgenogram of the thorax in coarctation of the thoracic aorta. Note the bilateral rib notching and also the site of coarctation at the level of the sixth intercostal space posteriorly on the left. (*b*) Posteroanterior roentgenogram of the thorax in coarctation of the thoracic aorta. Note the notched margin of the descending portion of the arch at the level of the left fifth intercostal space posteriorly. This notch indicates the site of coarctation. Evidence of rib notching in this case is minimal and could very easily be missed.

children, in whom notching of the ribs as yet may not have developed.

Roentgenoscopy at times may very accurately depict the site of coarctation and the degree of poststenotic dilatation of the aorta, these changes being reflected in corresponding deformities of the barium-filled esophagus.

7. Right Aortic Arch. This anomaly usually is evident in the routine roentgenogram, and as a rule is not clinically significant. However, it may occasionally be significant when the condition of congenital vascular ring is present, and the surgeon who performs a Blalock operation is interested in the side of the aorta. In infants and children it may be difficult to demonstrate. If the tracheal shadow shows an indentation on the right (Fig. 18) and not on the left, at the level where the aortic arch should be, this is positive proof that the aorta arches on the right. The presence of a

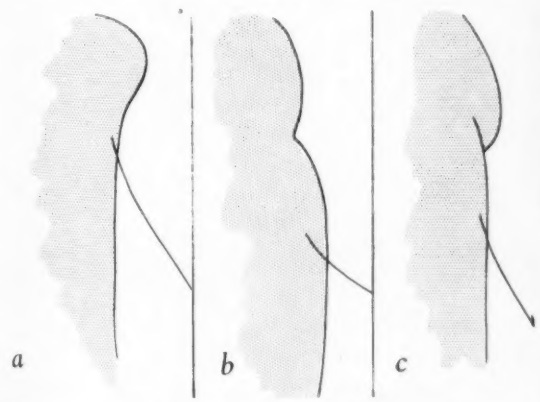

Fig. 17. (*a*) Normal aorta. (*b* and *c*) Two diagrammatic examples of the type of aortic notching seen in coarctation of the aorta.

Fig. 18. Right aortic arch indenting trachea on the right.

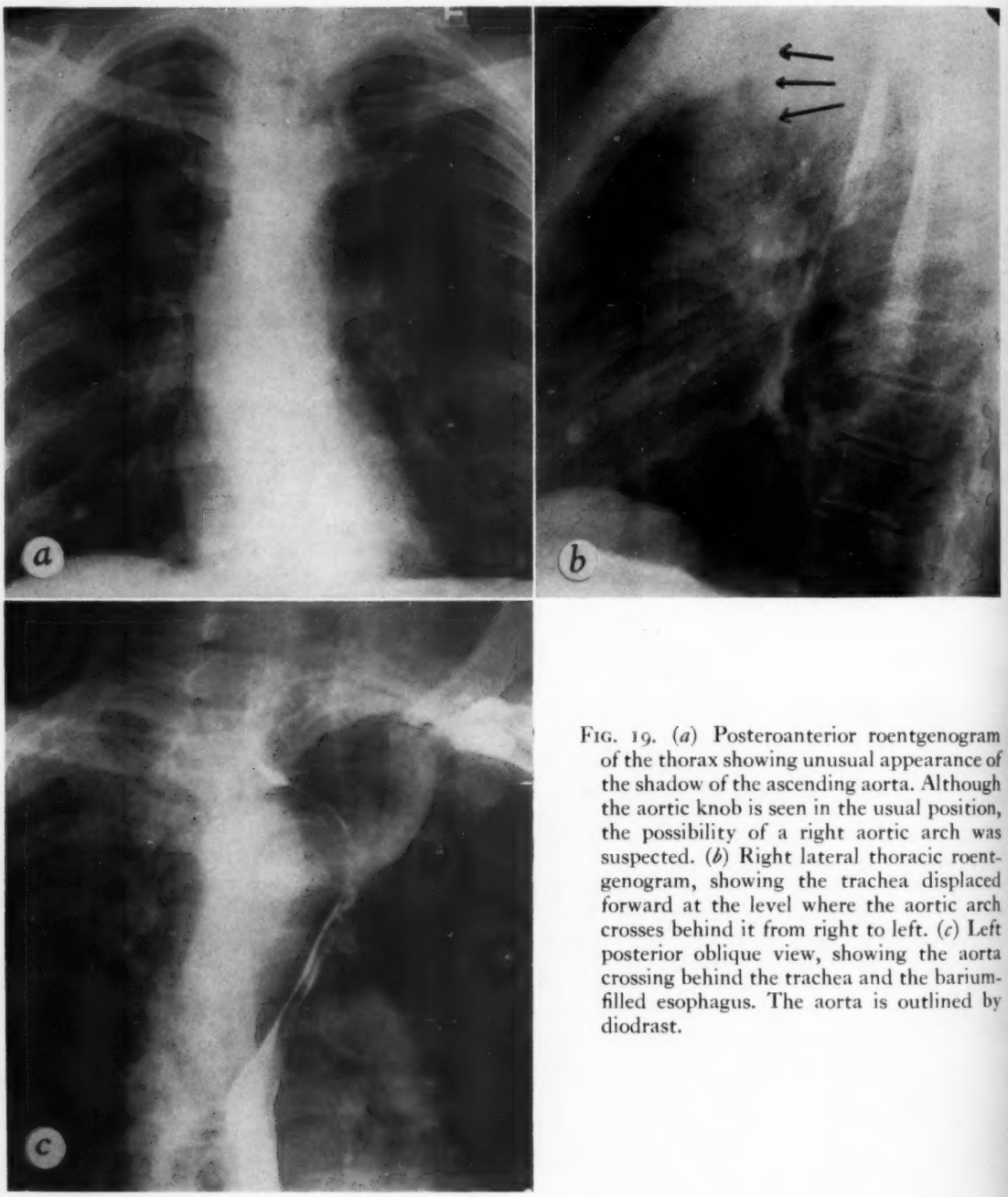

Fig. 19. (*a*) Posteroanterior roentgenogram of the thorax showing unusual appearance of the shadow of the ascending aorta. Although the aortic knob is seen in the usual position, the possibility of a right aortic arch was suspected. (*b*) Right lateral thoracic roentgenogram, showing the trachea displaced forward at the level where the aortic arch crosses behind it from right to left. (*c*) Left posterior oblique view, showing the aorta crossing behind the trachea and the barium-filled esophagus. The aorta is outlined by diodrast.

vascular ring can be demonstrated, in many cases, by the deformity produced on the esophageal shadows as seen during the swallowing of barium, and occasionally the plain roentgenograms will show a bilateral indentation on the trachea.

In the adult person, the right aortic arch may simulate a mediastinal mass in the posteroanterior roentgenogram, and the swallowing of barium may be extremely valuable in elucidating the true situation (Fig. 19, *a*, *b* and *c*).

8. Anomalous Venous Connection of the Lungs. Certain types of anomalous pulmonary venous connection have strikingly characteristic configurations in the postero-

anterior thoracic roentgenogram. On the other hand, anomalous pulmonary venous connection may be present without revealing any evidence of the anomalous vein or veins in the roentgenogram. Two signs that may be seen in anomalous pulmonary venous connection are the actual anomalous vein, and an unusual shift of the heart into the right hemithorax, sometimes giving the appearance of dextrocardia.

In our experience the most common type of anomalous pulmonary venous connection has been that in which at least a part of the right lung drains into the superior vena cava or high into the right atrium. In not one of 12 such cases was there any evidence of the presence of an abnormal vein, and in only 1 of the 12 was there an unusual degree of bulging of the right cardiac silhouette into the right hemithorax.

In 1 case in which the right lung drained to a persistent left superior vena cava, no direct evidence of the anomalous vein could be detected. In 4 cases, all the pulmonary veins drained either into the coronary sinus or into the coronary sinus and right atrium. In 2 of these the only change apparent in the thoracic roentgenograms was marked increase in pulmonary vascularity, and in the other 2 cases there was also some degree of bulging of the region of the right atrium into the right hemithorax.

The type of total anomalous venous connection in which the lungs drain into the left innominate vein via a partially persistent left superior vena cava (left vertical vein), resulting in the so-called figure-of-eight configuration of the heart and superior mediastinal shadows, appears to result in an extremely characteristic roentgenographic image (Fig. 20, *a*, *b* and *c* and 21, *a*, *b* and *c*). It is so characteristic that, if the evidence of increased vascularity of the pulmonary vessels also is present, the diag-

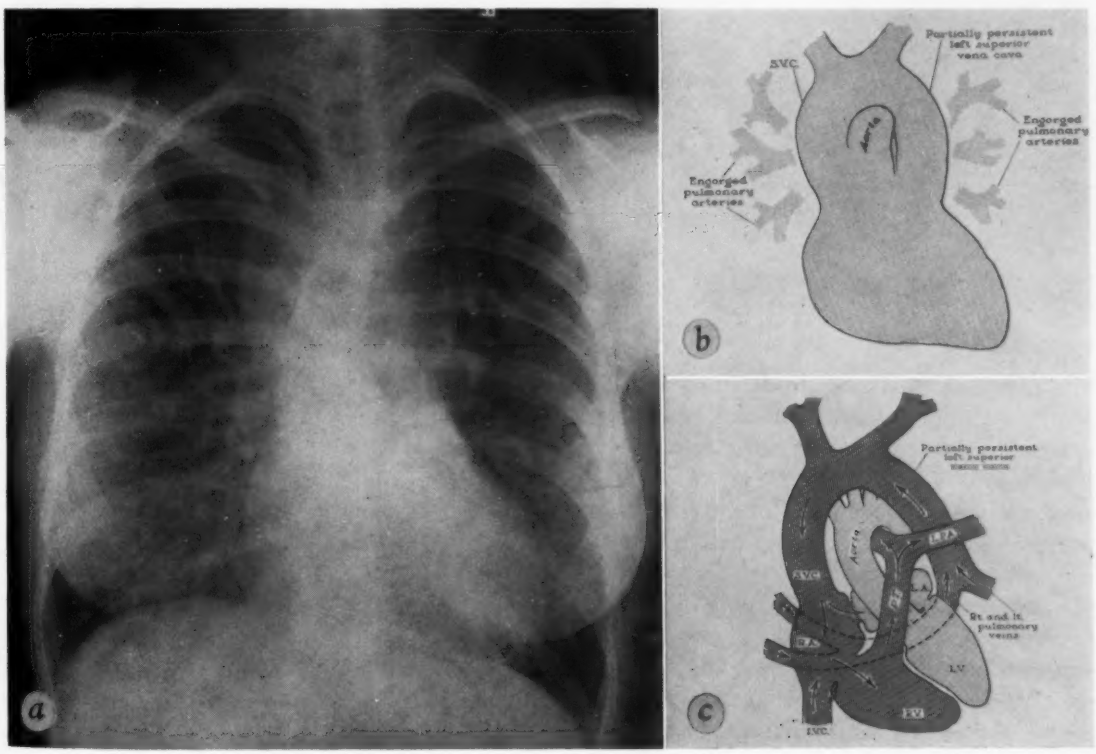

Fig. 20. (*a*) Posteroanterior roentgenogram of the thorax showing characteristic contour seen in the figure-of-eight type of total anomalous pulmonary venous connection. Diagrams *b* and *c* explain the circulatory anomaly in this type of total anomalous pulmonary venous connection.

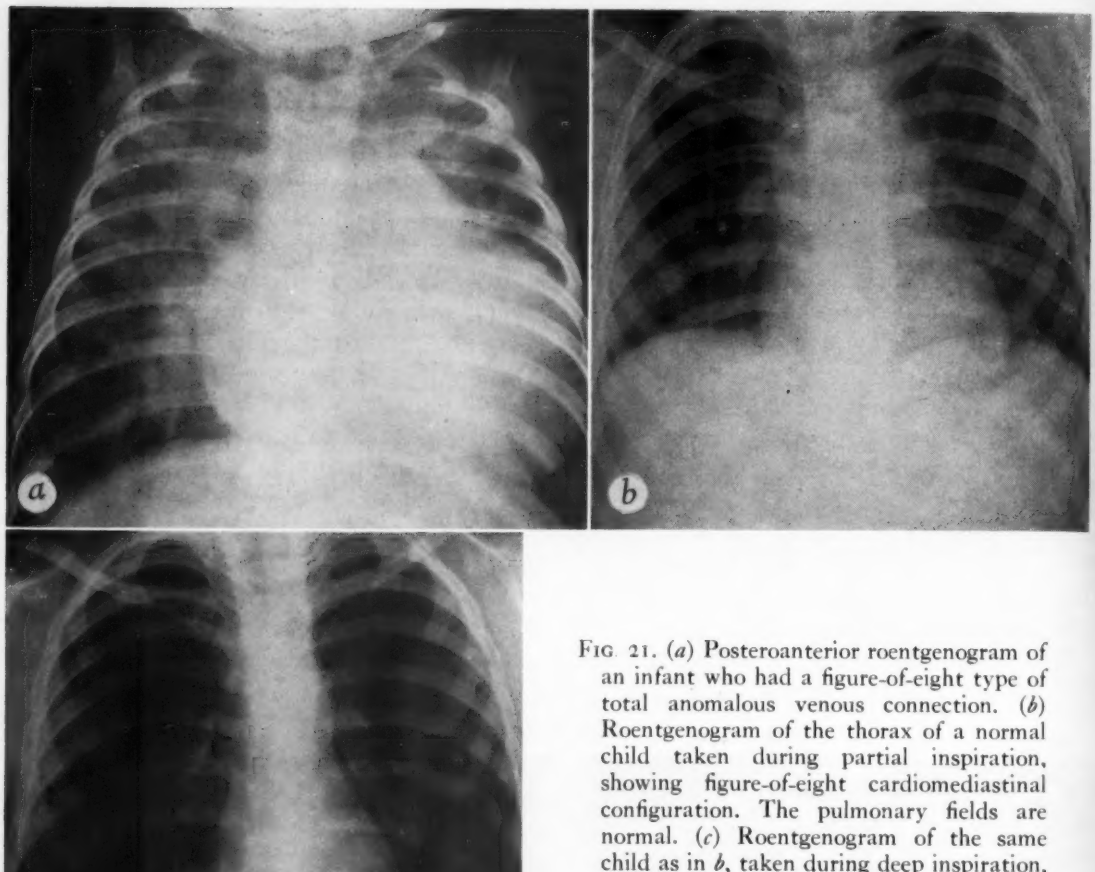

Fig. 21. (*a*) Posteroanterior roentgenogram of an infant who had a figure-of-eight type of total anomalous venous connection. (*b*) Roentgenogram of the thorax of a normal child taken during partial inspiration, showing figure-of-eight cardiomediastinal configuration. The pulmonary fields are normal. (*c*) Roentgenogram of the same child as in *b*, taken during deep inspiration, showing normal cardiomediastinal configuration.

nosis probably can be made unequivocally on the basis of a posteroanterior roentgenogram. We have noted this appearance in 5 patients. In 4 it was proved by catheterization and in 1 the anomalous vessels were noted during a thoracic exploratory operation. In a baby of four months the anatomic abnormality was present, but the thoracic roentgenogram did not reveal the widened superior mediastinal shadow, and this accords with Keith's observation that the figure-of-eight shadow usually is not seen before the age of eight months.

Another anomaly of pulmonary venous drainage that presents a characteristic roentgenologic appearance is found in patients in whom all or a large part of the right lung drains into the inferior vena cava (Fig. 22, *a* and *b* and 23). The anomalous pulmonary vein is seen, in the posteroanterior roentgenogram, to course downward in the right paracardiac region, disappearing in the shadow of the right hemidiaphragm. We have noted this shadow in 5 patients, in 2 of whom the condition was proved by cardiac catheterization. In only 1 of the 5 was the condition accompanied by evidence of another cardiovascular anomaly, namely, an atrial septal defect. In 2 of the cases the right hemithorax appeared

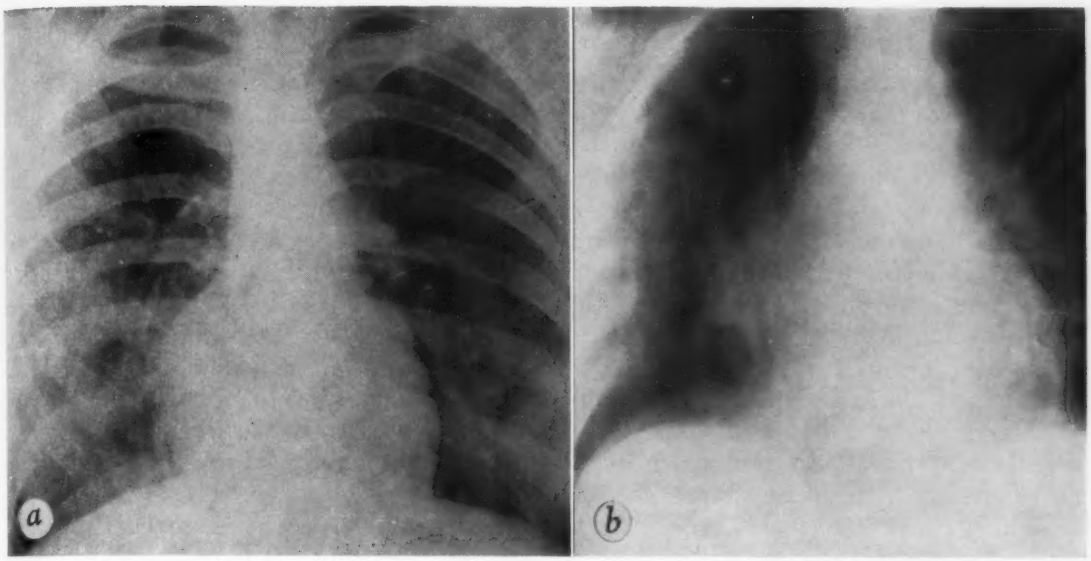

Fig. 22. (*a*) Posteroanterior roentgenogram of the thorax, showing bulge of the border of the right side of the heart into the right side of the thorax and a peculiar "streaky" appearance of the vessels along the right side of the heart. These features should always arouse the suspicion of anomalous venous connection of the right lung. (*b*) Laminagram of the right base demonstrates the anomalous pulmonary vein joining the cardiovascular shadow in the region of the inferior vena cava.

smaller than the left, and when these patients underwent bronchography it was demonstrated that each had agenesis of a part of the right lung. This probably accounts for the fact the the right hemithorax was smaller than the left, and suggests a relationship of this condition to bronchopulmonary sequestration. Although patients with anomalous drainage of the right lung, unassociated with other cardiovascular anomalies, have no specific cardiovascular symptoms, the condition has a certain practical importance in that the anomalously draining lung contributes much less to oxygenation of the blood than does the left lung, and is essentially functionless so far as preservation of life is concerned, so that if any disease occurs in the normal left lung, the patient may be in grave danger.

Acquired Heart Disease. 1. Mitral Valve Disease. From the roentgenologic point of view (the posteroanterior thoracic roentgenogram), mitral heart disease may have some similarity to the picture of atrial or ventricular septal defect: the enlarged pulmonary trunk, the evidence of enlargement of the right side of the heart and the engorged hilar vessels. A comparative study was therefore made of 73 cases in which mitral stenosis was proved at operation.

The transverse size of the heart (cardiothoracic ratio) is of no assistance in the

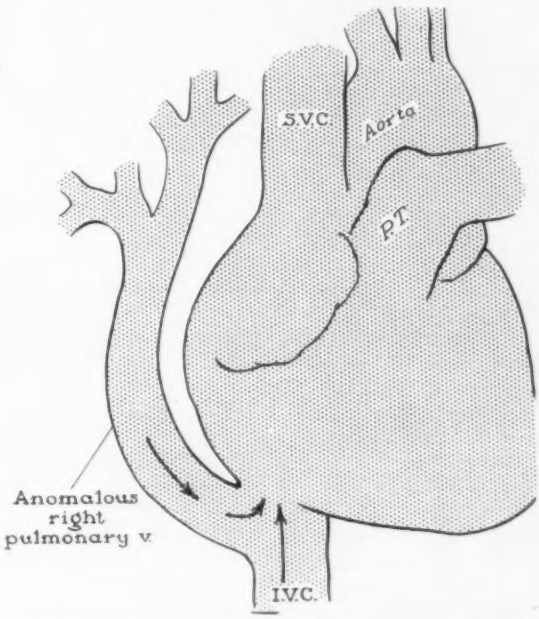

Fig. 23. Diagram illustrating the pulmonary venous anomaly seen in Figure 22.

diagnosis of mitral heart disease. In the 73 cases of mitral stenosis evaluated, the cardiothoracic ratio was slightly more than or slightly less than 50 per cent in the great majority of cases.

Pulmonary Trunk. In 37 per cent of cases, the shadow of the pulmonary trunk appeared to be normal. In 30 per cent of cases it was slightly enlarged, in 29 per cent it was moderately enlarged, and in 4 per cent it was markedly enlarged.

Aorta. It has been stated that the aortic knob usually is small in the presence of mitral heart disease. This is not so. The shadow of the aortic knob was graded as hypoplastic in only 10 per cent of cases, and as small in 17 per cent of cases. In the other 73 per cent of cases it was normal in appearance. This feature is of no differential value in distinguishing regurgitation from stenosis.

Vascularity of Pulmonary Fields. In 69 per cent of cases the appearance of the vascular branches in the pulmonary fields was normal. In many of these cases, however, there was engorgement of the hilar vessels, best seen on the right side. However, in this situation the engorgement has a different appearance from that seen in atrial or ventricular septal defect with pulmonary hypertension associated with obliterative pulmonary arteriolar changes (Fig. 24, *a* and *b*). In the latter two conditions, the outlines of the engorged arteries usually are well seen, whereas in mitral stenosis the right hilar shadow is seen to show a hazy amorphous enlargement, individual vessels not showing; the appearance occasionally resembles that of hilar adenopathy. This probably is caused by several factors, such as a concomitant haziness of the entire pulmonary field due to a variable amount of deposition of hemosiderin, engorgement of the pulmonary lymphatic vessels, and distention of the pulmonary veins as well as the pulmonary arteries (there is hypertension of both of

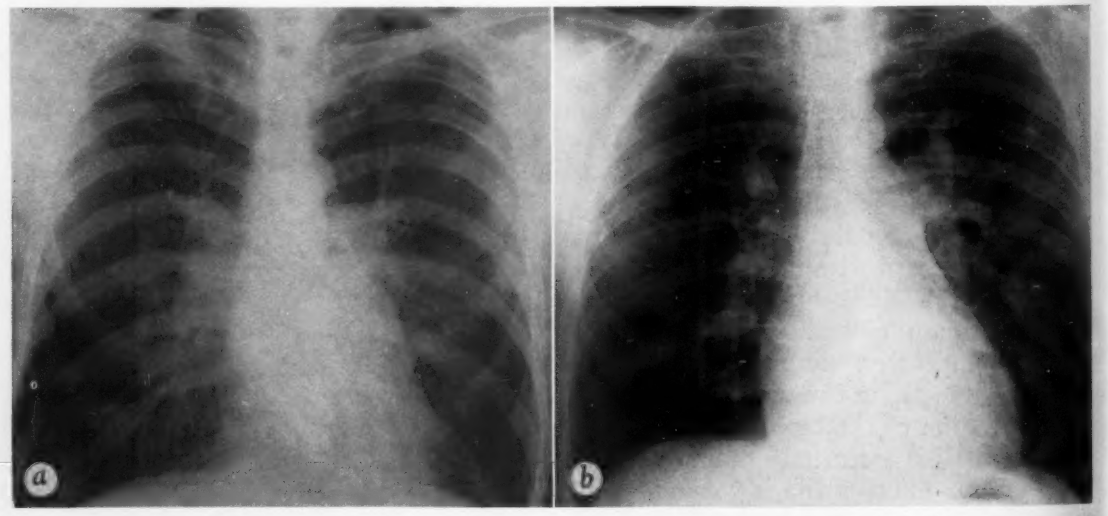

Fig. 24. (*a*) Posteroanterior view in mitral stenosis. Many of the features of the mitral stenotic heart are seen in this roentgenogram: the prominent and congested hilar vessels; the prominence of the left auricular appendage just below the left hilus; the double contour on the right due to enlargement of the left atrium; the costophrenic lymphatic lines; some evidence of hemosiderosis. Note that the right paracardiac area below the hilus is hazy as compared with the appearance of this region in septal defect with pulmonary hypertension (see text). (*b*) Posteroanterior roentgenogram of the thorax of a patient with an atrial septal defect and fairly marked pulmonary hypertension. In such cases the hilar arteries are engorged, but because the pulmonary hypertension is arterial only and not veno-arterial, as in mitral stenosis, the pulmonary veins and lymphatic vessels are not engorged. Therefore, the lungs appear clearer than in mitral heart disease and the right paracardiac area is clear as compared to the appearance in Figure 24*a* (see text). See also Figures 5 and 6.

these systems in mitral stenosis). Furthermore, the engorged pulmonary veins cross medially just below the pulmonary arteries and therefore tend to obscure the outlines of the latter. In the presence of septal defects accompanied by obliterative arteriolar changes the right paracardiac region, below the hilus, tends to be clear because the pulmonary veins often are not engorged under increased tension, since the obstruction to pulmonary blood flow lies on the proximal side of the capillary bed. In the presence of mitral stenosis this area is obscured by a combination of engorged veins and arteries, the obstruction lying beyond the pulmonary veins.

Roentgenoscopy. The pulsations of the hilar vessels were graded as normal in 94 per cent of cases of mitral stenosis, whereas there was reported to be slight increase in only 3 per cent and a moderate increase in another 3 per cent. Of course, roentgenoscopy almost invariably will reveal some enlargement of the left atrium on the swallowing of barium. This is a very reliable roentgenoscopic sign, although in itself is not specific for rheumatic mitral heart disease.

Once again, it should be stressed that evaluation of ventricular size during roentgenoscopy often is more unreliable than not. The enlargement of the right ventricle usually can be noted accurately enough on the posteroanterior or lateral thoracic roentgenogram. Evaluation of the size of the left ventricle by roentgenoscopy can be complicated by posterior displacement of the heart and by small pericardial effusions. Roentgenoscopy is too inaccurate to be of value unless the left ventricular enlargement is marked.

A roentgenoscopic sign in mitral regurgitation is systolic expansion of the left atrium. This finding, in the oblique projections, is too inaccurate to be valuable. In these projections, the barium-filled esophagus may show motions caused by pulsations of the descending aorta which may be confusing. Moreover, it is possible that diastolic filling of the left atrium may be confused with systolic expansion. It is probably better for the roentgenologist to judge systolic expansion of the left auricular appendage in the posteroanterior projection. Moreover, even if it is seen to be present, the final decision should be clinical when the matter of operation is in question.

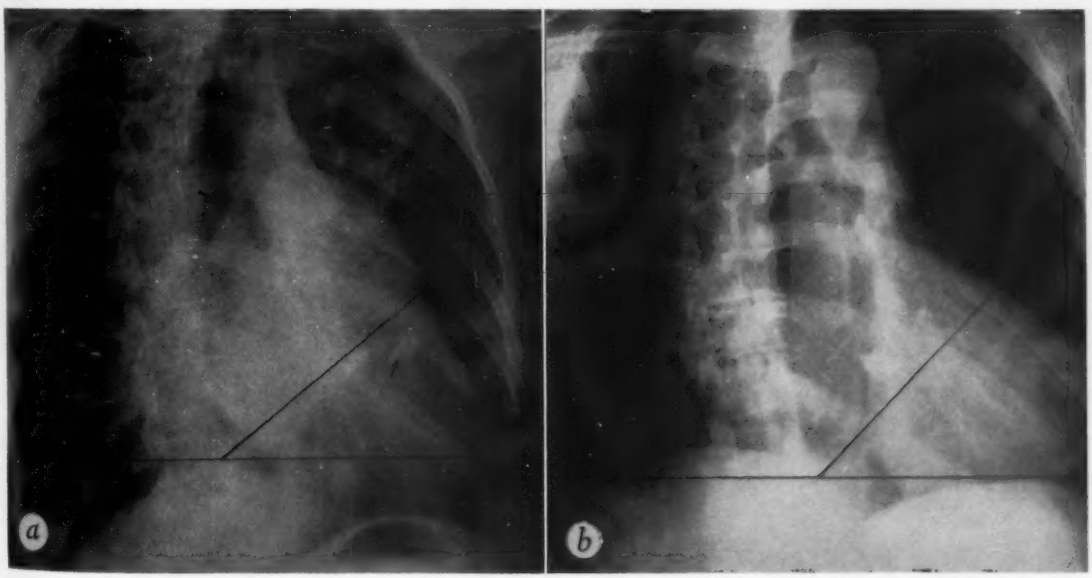

Fig. 25. (*a*) Mitral heart disease in right anterior oblique projection. The calcified mitral valve lies to the left of a line drawn through the point of opposite pulsations and at about 45 degrees to a horizontal plane. (*b*) Aortic valvular disease with the calcified aortic valve lying to the right of the line.

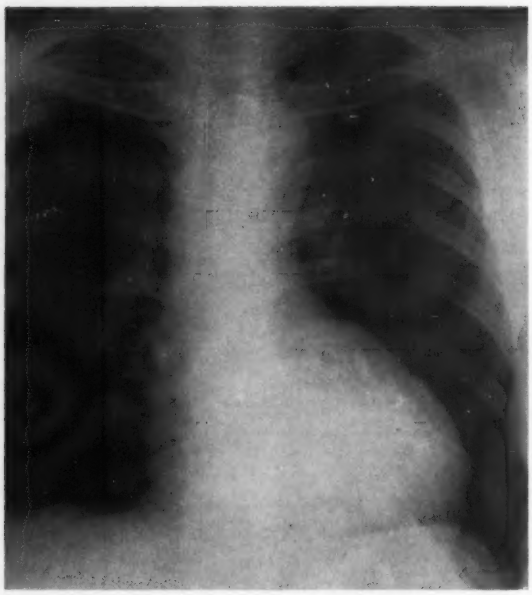

Fig. 26. This posteroanterior configuration is that of left ventricular enlargement. This picture shows the deepened cardiac waist and accentuation of the left ventricular curve. The pulmonary trunk and hilar vessels are normal. This roentgenogram is from a patient with aortic stenosis. Roentgenoscopy in such a case will contribute nothing to evaluating left ventricular enlargement, but should be of value in the investigation of the vigor of pulsations in the aorta (tends to be quiet in stenosis, active in regurgitation); calcification of the cardiac valves; and possible left atrial enlargement (this may occur in aortic stenosis when the left ventricle is failing).

Another definite place for roentgenoscopy is determination of the presence of valvular calcification in heart disease. The maneuvers recommended by Sosman have been found valuable (Fig. 25, *a* and *b*). However, they probably are most nearly accurate in cases in which either the mitral valve or the aortic valve is diseased; that is, when the configuration of the heart is predominantly aortic or mitral. The proximity of these valves to each other would complicate separate identification when both valves are diseased and calcified. It is likely that identification will be facilitated by the use of image intensification.

2. Aortic Valvular Disease. An evaluation of the configuration of the border of the left side of the heart in the posteroanterior film often will enable the examiner to judge whether ventricular enlargement is primarily left ventricular or right ventricular in type. It is when both ventricles are enlarged that interpretation becomes diffi-

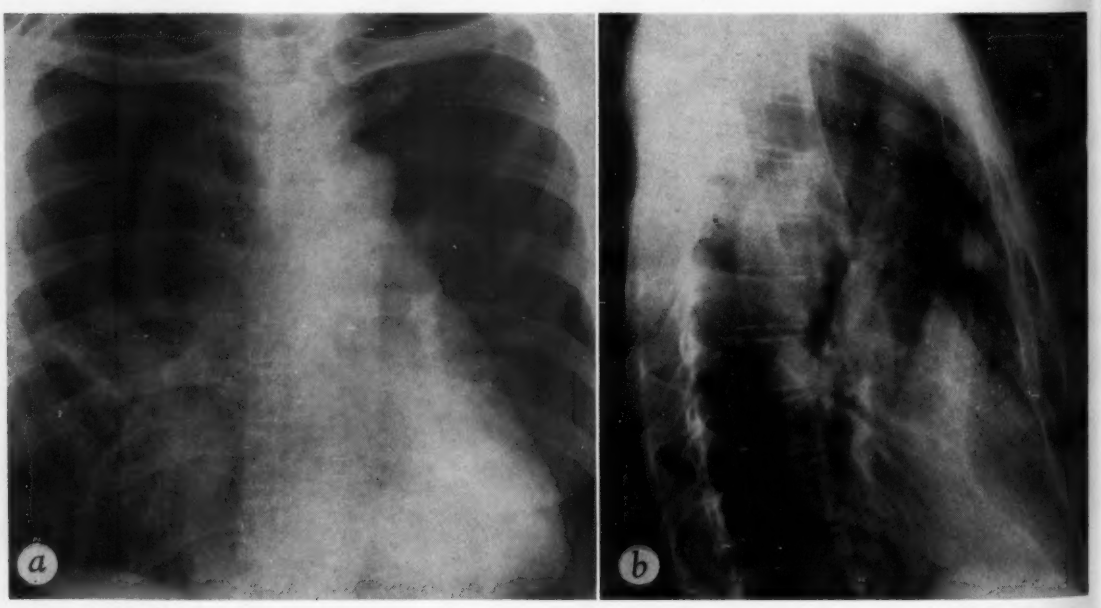

Fig. 27. (*a*) Posteroanterior roentgenogram of patient with marked funnel-chest deformity. Note appearances simulating consolidation of the middle pulmonary lobe and "straightening" of the border of the left side of the heart. Note also the clarity with which the lower part of the thoracic section of the spinal column can be seen. (*b*) Lateral view of same patient.

cult. But again, the posteroanterior thoracic roentgenogram probably will be at least as reliable as roentgenoscopy in such cases, and may even be considerably more reliable. Thus, when the right ventricle is predominantly enlarged, the border of the left side of the heart tends to lose the shallow, gentle S-curve of the normal, and tends to straighten out and subsequently assume a variable, convex curve.

On the other hand, predominant enlargement of the left ventricle will lead to greater prominence of the lower left cardiac bulge, and accentuation of the lower curve of the S-configuration and deepening of the cardiac waist. If the aortic valve is incompetent, the curve of the ascending aorta tends to become more accentuated, both curves of the S-configuration therefore being accentuated (Fig. 26).

Naturally, the build of the patient and the possibility of a transverse position

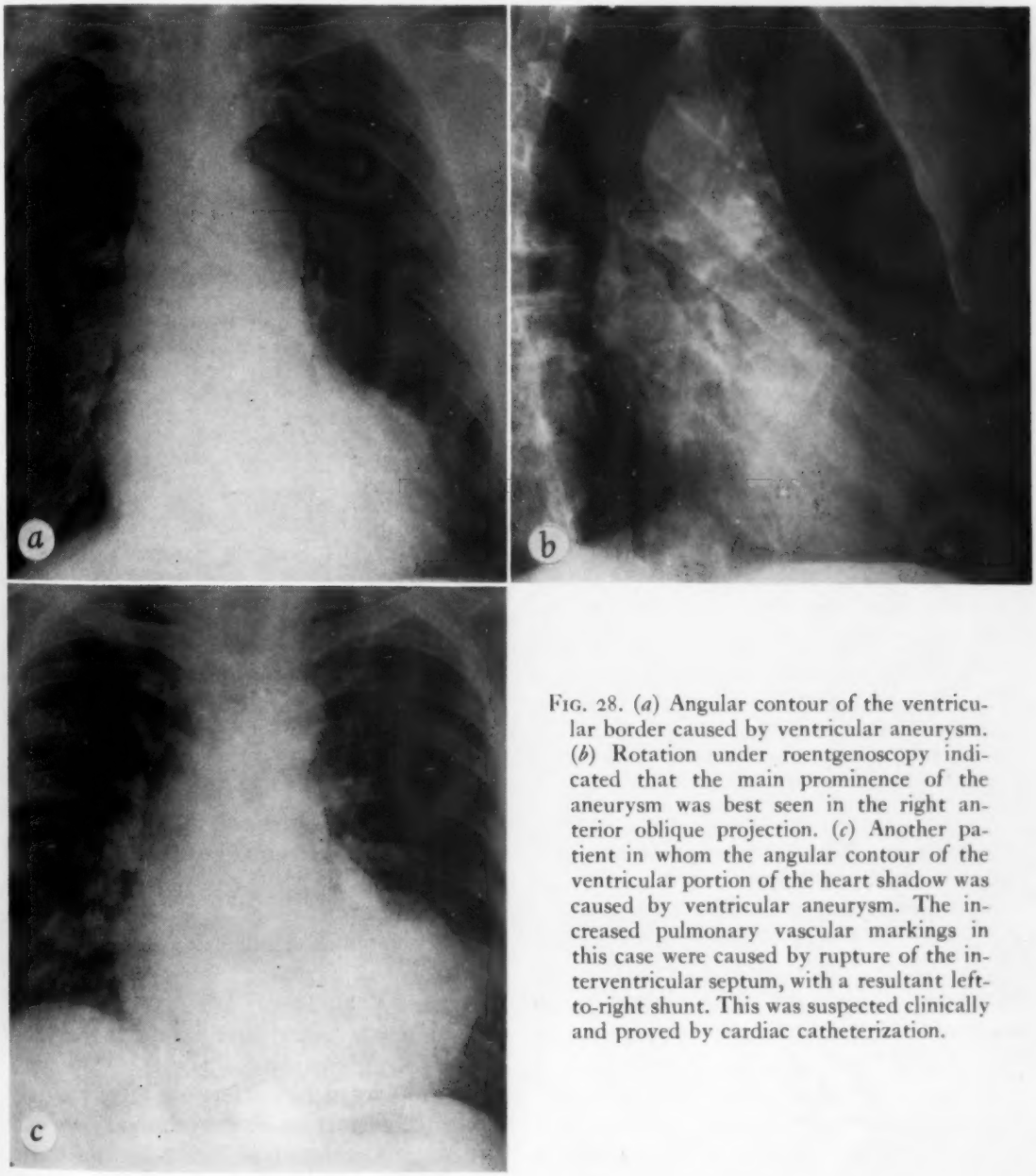

Fig. 28. (*a*) Angular contour of the ventricular border caused by ventricular aneurysm. (*b*) Rotation under roentgenoscopy indicated that the main prominence of the aneurysm was best seen in the right anterior oblique projection. (*c*) Another patient in whom the angular contour of the ventricular portion of the heart shadow was caused by ventricular aneurysm. The increased pulmonary vascular markings in this case were caused by rupture of the interventricular septum, with a resultant left-to-right shunt. This was suspected clinically and proved by cardiac catheterization.

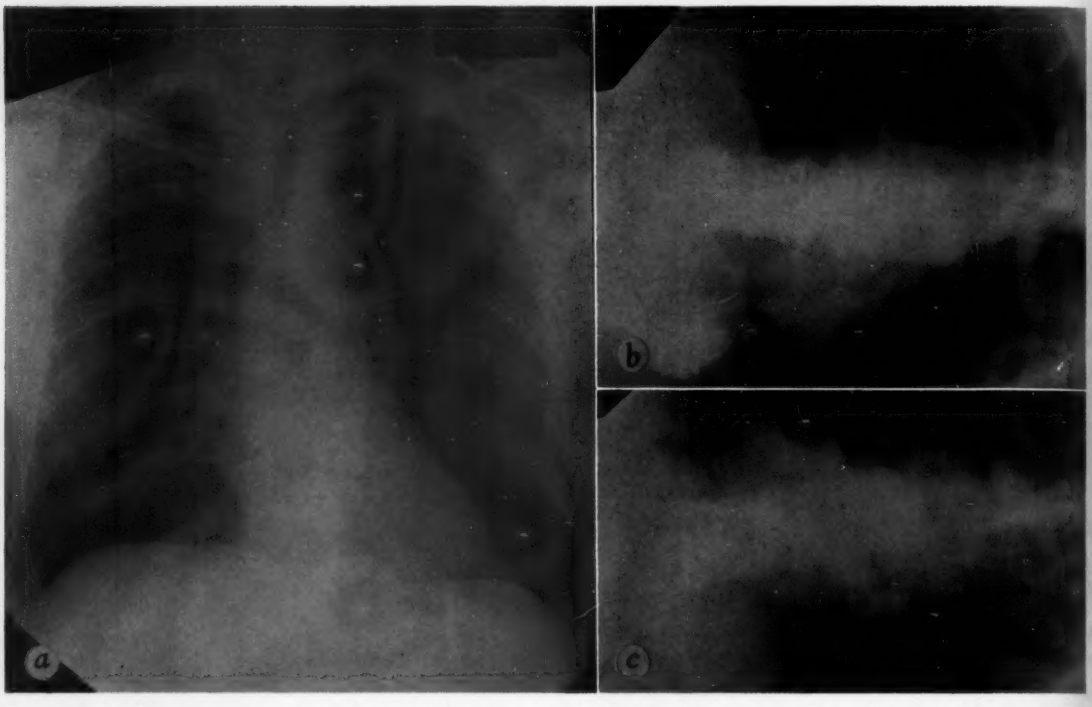

Fig. 29. (*a*) Abnormal contour to the lower border of the right side of the heart, the location of the abnormality having been confirmed by roentgenoscopy. (*b*) A left lateral decubitus view, showing the "mass" to be smaller than it is in *c*, which is a right lateral decubitus view. In the latter view the lesion is rounder and fuller. The difference in appearance between the two decubitus views indicates a fluid-containing lesion; this proved to be a pericardial diverticulum, that is, a pericardial cyst communicating with the pericardial cavity.

of the heart should be considered. But it is practical to point out that the roentgenologist does not function in a void, and when the condition at hand is questionable, it is natural that he accept the limitations of his method and abide by the often more nearly accurate methods of evaluation at the disposal of his clinical colleagues.

The pulmonary trunk in aortic valvular disease is normal. The aortic knob usually is normal, but because of an increased prominence of the curve of the aortic arch, especially when aortic regurgitation is present, the aortic knob may be more prominent than normally seen in roentgenograms. The vascular structures in the pulmonary fields show no abnormality unless heart failure causes some hilar congestion. Pulmonary arterial pulsations are normal, but the activity of the aortic arch tends to indicate the type of lesion, a very active pulsation of the arch suggesting aortic regurgitation rather than stenosis.

An interesting finding in certain cases of aortic valvular disease, without mitral valvular disease, is the occasional occurrence of left atrial enlargement demonstrated both in the posteroanterior roentgenogram and following the swallowing of barium. This is indicative of failure, and in our cases in which catheterization has been done, left atrial enlargement always has been accompanied by an increased pulmonary-wedge pressure. Furthermore, costophrenic septal lines may be present in such cases, being additional indications of an increased pulmonary venous pressure. These lines, which probably represent engorged lymphatic vessels, are much more commonly seen in mitral heart disease than in aortic valvular disease. But when they do occur in the presence of heart disease, they are an extremely accurate indication that pulmonary venous pressures are elevated.

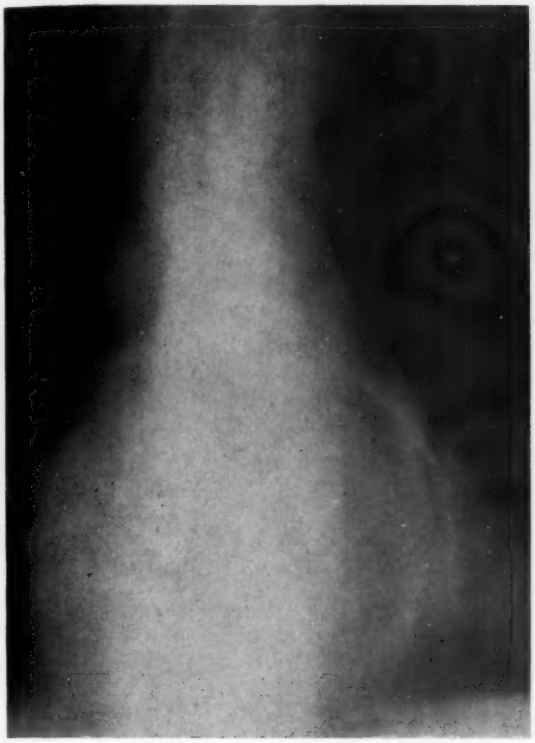

Fig. 30. Pericardial calcification was seen on the routine posteroanterior roentgenogram of the thorax, and laminagraphy was used to delineate the lesion a little better. One section is shown here.

ADDITIONAL NOTES

A few brief notes are appended on a number of cardiovascular conditions in which posteroanterior roentgenograms of the thorax can either provide a positive diagnosis or guide the examiner to the correct ultimate diagnosis.

1. Funnel Chest. This is depicted in Figure 27, *a* and *b*. In the patient who has a fairly marked funnel-chest deformity, the heart may be displaced and rotated into the left hemithorax so as to simulate the so-called straight left border of mitral heart disease. Commonly, roentgenograms of these patients also will give an appearance suggesting middle-lobe pulmonary consolidation. A characteristic feature of the roentgenogram, suggesting that a funnel-chest deformity is present, is the rather clear visualization of the lower part of the thoracic section of the spinal column in the posteroanterior view. This part of the spinal column is much more clearly visualized in the presence of funnel-chest deformity than it is when the heart is normally placed. A lateral roentgenogram, or if possible, examination of the patient's thorax, will settle the point.

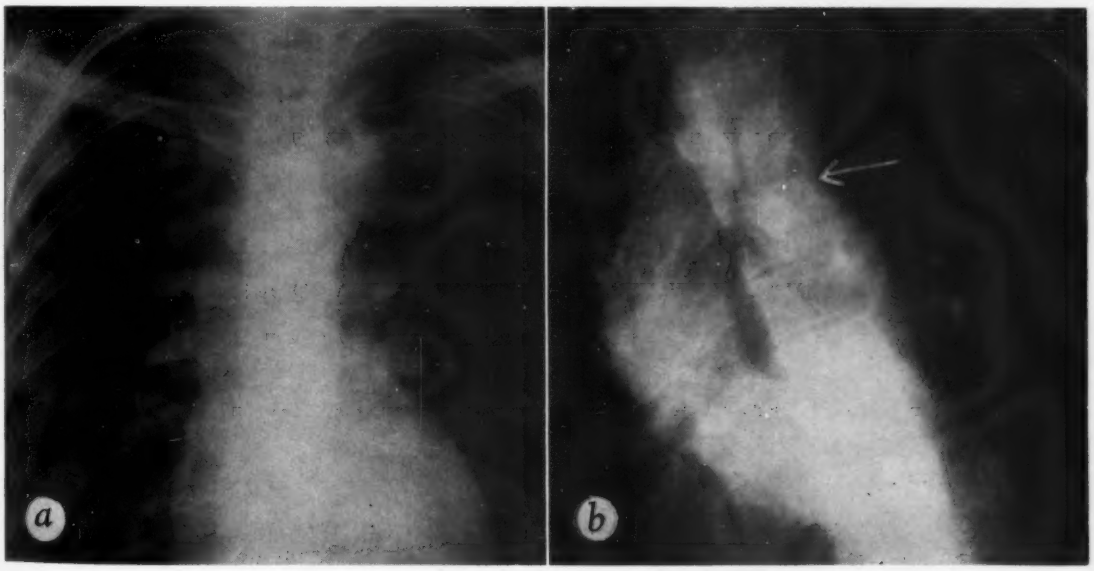

Fig. 31. (*a*) Posteroanterior roentgenogram of the thorax shows a widening of the superior mediastinal shadow on the left. The possibility that this was a mass or a vascular lesion was raised. Roentgenoscopy was noncontributory. (*b*) An angiocardiogram was done; it revealed the aorta to be buckled in the region of the ligamentum. The appearance of the aortic contour at this site suggests coarctation, but the lumen of the aorta is wide and there was no clinical indication of coarctation.

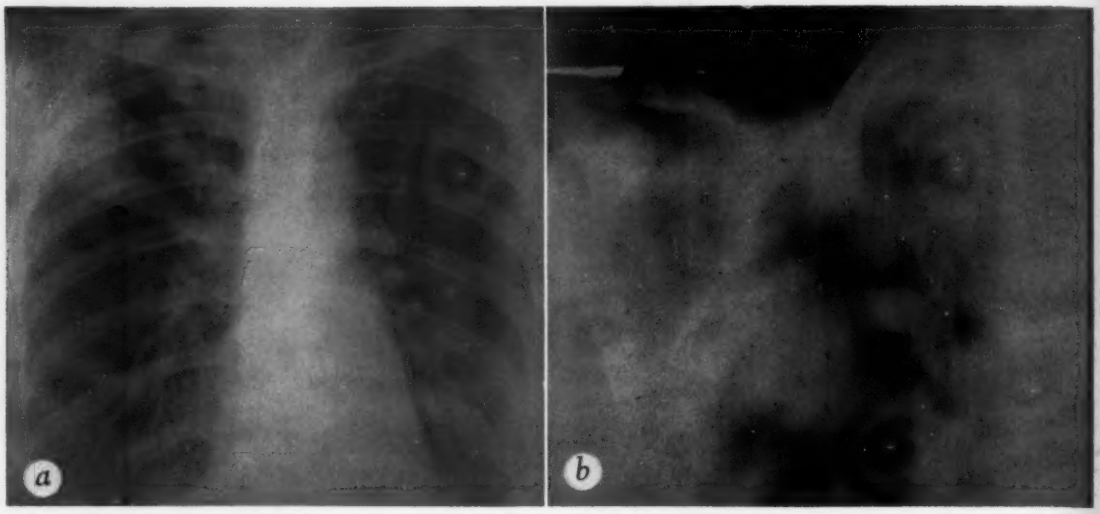

Fig. 32. (*a*) Rounded density in the upper lobe of the right lung. The density is connected to the hilar region by the two large vascular trunks. (*b*) Laminagram of the same region, clearly delineating the arteriovenous malformation with its artery of supply and efferent vein.

In summary, then, the features of the posteroanterior roentgenogram suggesting funnel chest are: straightening of the border of the left side of the heart, an appearance simulating consolidation of the middle lobe of the lung, and the rather clear visualization of the lower part of the thoracic section of the spinal column.

2. Cardiac Aneurysm. The contours of the heart, whether normal or enlarged, are characteristically smooth. Any variation from the smoothly rounded contours, whether the heart is normal in size or large, should be regarded as abnormal. One condition which may show an interruption of the smooth ventricular outline is ventricular aneurysm which follows cardiac infarction (Fig. 28, *a*, *b* and *c*). When roentgenoscopy is done in such a case, the involved region either will not pulsate or will show paradoxic pulsation.

3. Pericardial Cyst. This benign lesion may cause an abrupt interruption of the cardiac contour (Fig. 29, *a*, *b* and *c*). It is a smooth but obviously abnormal interruption. Roentgenoscopy will not be of much value in distinguishing the lesion. But right-and-left lateral decubitus roentgenograms often will show sufficient alteration in the contour of the mass to make it obvious that a fluid-containing lesion is present, and that in all likelihood the lesion is a pericardial cyst.

4. Calcified Pericardium. When a well-exposed posteroanterior thoracic roentgenogram is made, there should be no difficulty

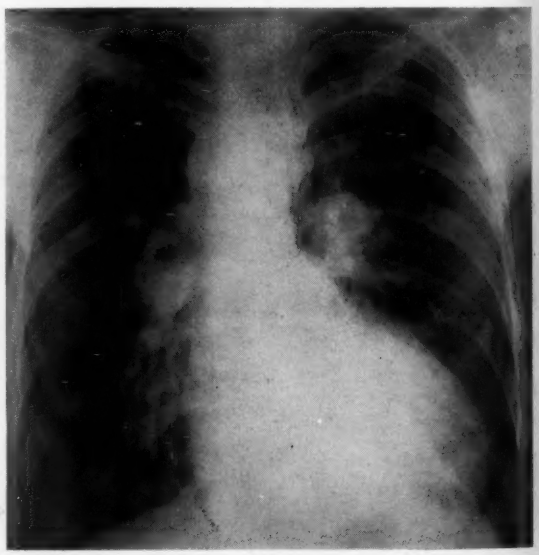

Fig. 33. Posteroanterior roentgenogram of the thorax, showing right "aortic" arch. Note that the left hilar vessels do not appear to have a normal connection with the outflow tract of the right ventricle via a pulmonary trunk. The hilar vessels actually are arising from a truncus.

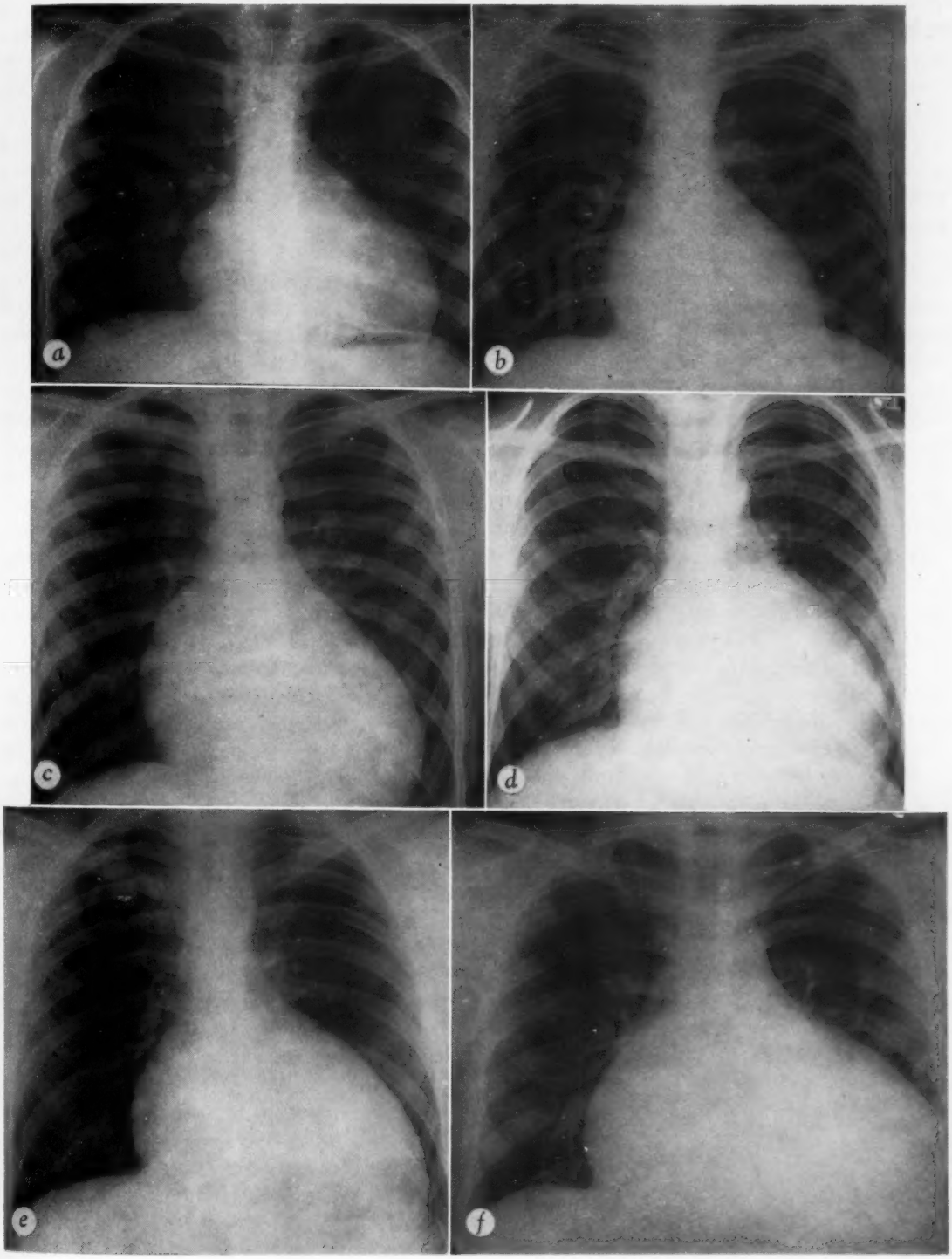

Fig. 34. (*a*, *b*, *c*, *d*, *e* and *f*) Posteroanterior views of 6 different patients with Ebstein's malformation of the tricuspid valve. Except for differences in size of the heart, the cardiac configurations in all these views show a considerable likeness.

in detecting calcification in the contour of the heart. Roentgenoscopy is valuable in determining the extent of the calcium. Laminagrams may provide a good record of this finding; an example is presented in Figure 30. Very rarely, the myocardium will calcify. When it does, the calcification tends to be limited to the distribution of a cardiac chamber, usually the left atrium.

5. Subclinical Coarctation. Occasionally, a short ductus or a short ligamentum arteriosum will cause a kink in the aorta, just beyond the origin of the left subclavian artery. This kink does not significantly compromise the internal lumen of the aorta, and it is for this reason that the term "subclinical coarctation" was used to describe the condition. It may perhaps be more appropriate to call "subclinical coarctation" a "buckling" or "kinking" of the distal part of the aortic arch.

The significance of the condition is that it resembles a mass situated in the upper part of the mediastinum on the left (Fig. 31, *a* and *b*), and patients with such a condition actually have received roentgen therapy before the true nature of the lesion was proved. The examiner should be suspicious of the condition when, in a patient who has no other symptoms or signs suggesting coarctation, the appearance of the descending aorta simulates the kink seen in true coarctation. A good left anterior oblique view may demonstrate the buckle in the upper part of the descending thoracic aorta, and a successful angiocardiogram will demonstrate it well.

6. Pulmonary Arteriovenous Fistula. Occasionally, the positive diagnosis of a pulmonary arteriovenous fistula can be made on the basis of the posteroanterior thoracic roentgenogram. This requires demonstration of a dense, rounded shadow in the lung, connected to the region of the hilar vessels by two vascular trunks (Fig. 32, *a* and *b*). Laminagraphy occasionally may demonstrate the lesion with remarkable clarity.

7. Truncus Arteriosus. In the type of truncus arteriosus in which the lungs are supplied by two large "pulmonary arteries" arising from the ascending portion of the truncus, the posteroanterior thoracic roentgenogram may have a characteristic appearance (Fig. 33). Here the pulmonary trunk and left pulmonary artery do not arise out of the region of the outflow track of the right ventricle; rather, the pulmonary artery going to the left lung has no obvious connection with a pulmonary trunk or with the region of the right ventricular outflow tract. Instead, the vessel appears to come from the aorta (truncus).

8. Ebstein's Malformation of the Tricuspid Valve. The posteroanterior thoracic roentgenograms of 6 patients with Ebstein's malformation of the tricuspid valve are shown in Figure 34. The only comment necessary concerns the remarkable similarity in configuration of the heart in at least 5 of these cases. All the patients were cyanotic. It is seen that the pulmonary trunk is small or normal, and that the pulmonary fields may be looked upon as being anemic.

Section of Roentgenology
Mayo Clinic and Mayo Foundation
Rochester, Minnesota

Vol. 76, No. 4

CARDIAC ROENTGEN-RAY PATTERNS IN RHEUMATIC HEART DISEASE*

CORRELATION OF ROENTGEN AND OPERATIVE FINDINGS IN 150 CONSECUTIVE CASES

I. A PRELIMINARY REPORT

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PHILADELPHIA, PENNSYLVANIA

THE purpose of this paper is to present roentgen-ray patterns for the diagnosis of rheumatic heart disease by analysis of the cardiovascular silhouette in the single posteroanterior view of the chest. A study of these patterns should facilitate the recognition of these cardiac lesions in routine and survey posteroanterior films. This preliminary report is limited to mitral and aortic valvular disease.

MATERIAL

The material consists of preoperative roentgen examinations of 150 consecutive cases. Only a short interval of time usually elapsed between roentgen study and cardiac surgery. These cases were operated upon at Doctors Hospital, Philadelphia, Pennsylvania, during 1952 and 1953 by Dr. Charles P. Bailey and his associates. In addition to the posteroanterior view, our technique included the left lateral and both oblique projections with barium in the esophagus. Since it has been found that the posteroanterior view provided the main criteria for diagnosis, this paper is limited to the evaluation of this projection only.

METHOD OF CODING

The posteroanterior roentgenogram of a normal chest (Fig. 1) depicting the silhouette of the heart and great vessels is divided into six zones, three on the left side and three on the right side. Starting on the left side from above downward, Zone 1 denotes the aortic knob, Zone 2 encompasses the cardiac waistline (which is normally slightly concave), and Zone 3 indicates the left ventricular border. The line of demarcation between Zones 2 and 3 is created by dividing in half the distance between the lower border of the aortic knob and the left cardiophrenic angle. Starting on the right side from above downward, Zone 4 is the ascending aorta. Zones 5 and 6 comprise the upper and lower halves of the right atrial border, which extends from the right cardiovascular indentation to the right cardiophrenic angle.

With this zoning, we have developed a coding system in which the changes from the normal cardiovascular silhouette are recorded as plus or minus for each numbered

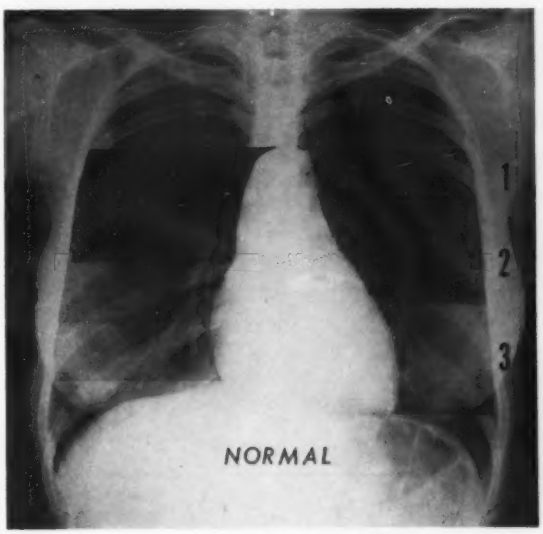

FIG. 1. Posteroanterior roentgenogram of a normal chest with the silhouette of the heart and great vessels divided into six zones.

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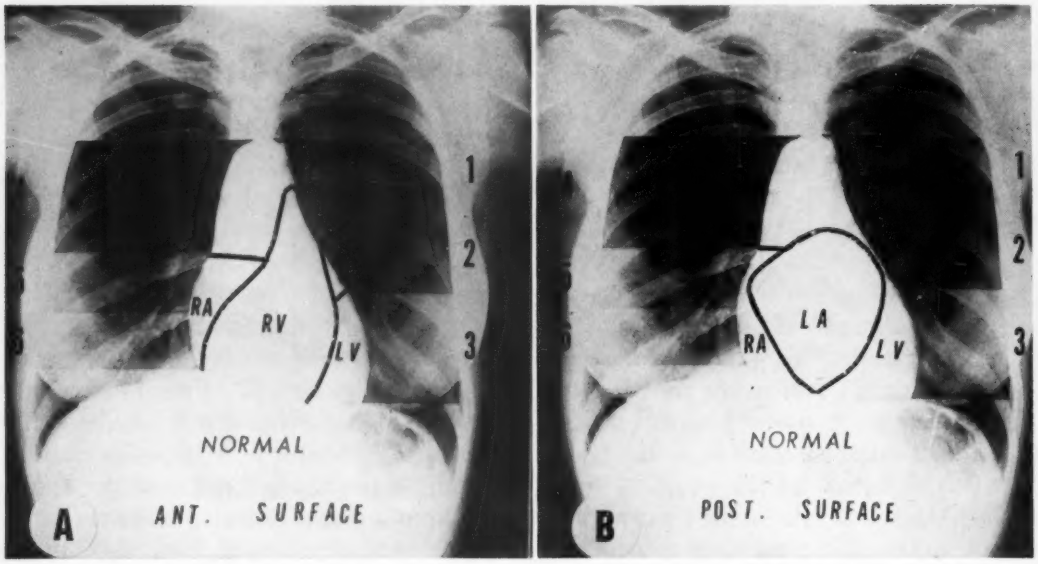


Fig. 2. Schematic representation of the chambers of the heart. On the left are the cardiac chambers as seen on the anterior surface of the heart (*A*), and on the right are the cardiac chambers as visualized through the posteroanterior projection on the posterior surface of the heart (*B*).

zone. When a plus or minus is encircled, it indicates an exaggerated change; when no change is present, no code number is assigned. The combination of these values is designated as the code for each case. After all cases are coded, characteristic patterns evolve for the various mitral and aortic valvular lesions. These patterns coincide with the surgical findings in a very large percentage of cases.

CARDIAC PATTERNS

Normal Cardiovascular Anatomy. A schematic representation of the chambers of the heart as they are viewed in a posteroanterior chest roentgenogram shows the cardiac chambers on the anterior surface of the heart and also as they appear on the posterior surface of the heart visualized through the posteroanterior projection (Fig. 2). In order to make a diagnosis of rheumatic heart disease, one must be able to demonstrate enlargement of the various chambers of the heart. In the normal heart, the cardiac borders in the posteroanterior view are formed by only two chambers, the left ventricular border on the left, designated as Zone 3, and the right atrium on the right, designated as Zones 5 and 6. In Figure 2*A* it is noted that the right ventricle, which is the anterior chamber, is not normally border forming. In disease the right ventricular enlargement produces a corresponding enlargement of the pulmonary artery-conus

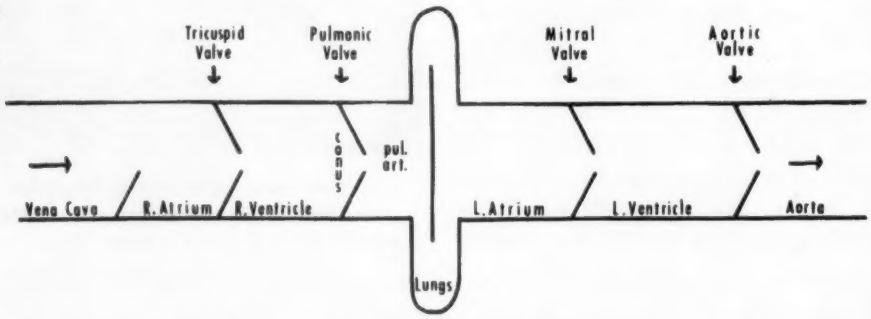


Fig. 3. Schematic drawing of the hemodynamics of the normal cardiovascular system.

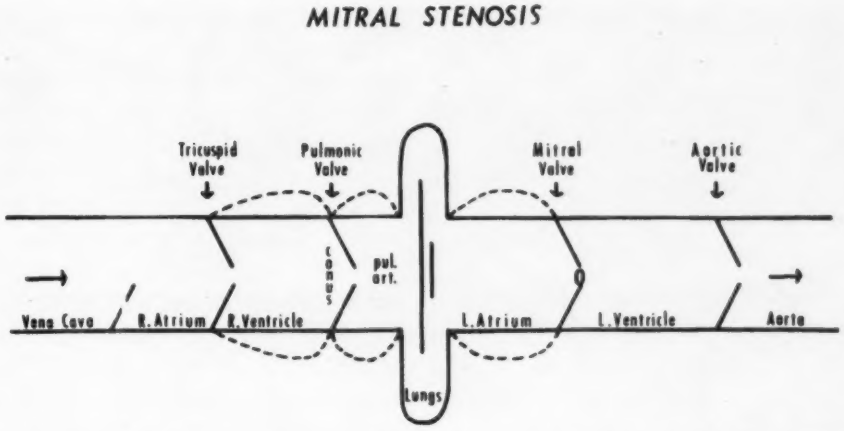


Fig. 4. Schematic drawing of the hemodynamics of mitral stenosis. Dotted lines indicate cardiac chamber enlargements. Attention is directed to the enlargement of the pulmonary artery-conus region which results in prominence of the cardiac waistline (Zone 2).

segment, which is reflected as a bulge in the waistline, designated as Zone 2. Similarly, in Figure 2*B* the left atrium, which is the posterior chamber, is not normally border forming. In disease the left atrium, in its enlargement, may overlap the upper right atrial border or create an increased density occupying Zone 5.

A schematic drawing (Fig. 3) demonstrates the hemodynamics of the normal cardiovascular system. The blood flows from the venae cavae to the right atrium. From the right atrium blood passes through the tricuspid valve into the right ventricle, and then through the pulmonic valve to the lungs. Proximal to the pulmonic valve is the pulmonary conus, and distal to the pulmonic valve is the pulmonary artery segment. From the lungs blood passes through the pulmonary veins into the left atrium. Blood courses from the left atrium through the mitral valve into the left ventricle, and then through the aortic valve, aorta and systemic circulation.

Mitral Stenosis. In mitral stenosis, partial obstruction occurs at the mitral valve due to narrowing of the orifice (Fig. 4). This causes dilatation of the left atrium, pulmonary vasculature, pulmonary artery-conus segment and right ventricle. This increased back pressure is contained within the muscular right ventricle and is not transmitted through the tricuspid valve.

In the posteroanterior roentgenogram of mitral stenosis (Fig. 5), we see the changes that occur as anticipated in the schematic drawing (Fig. 4). Zone 1, which is the aortic knob, is small. Zone 2, the left cardiac waistline, shows a bulge due to an enlarged pulmonary artery-conus segment on the basis of right ventricular enlargement. Zone 3, representing the left ventricular border, is usually not enlarged. Zone 4, which

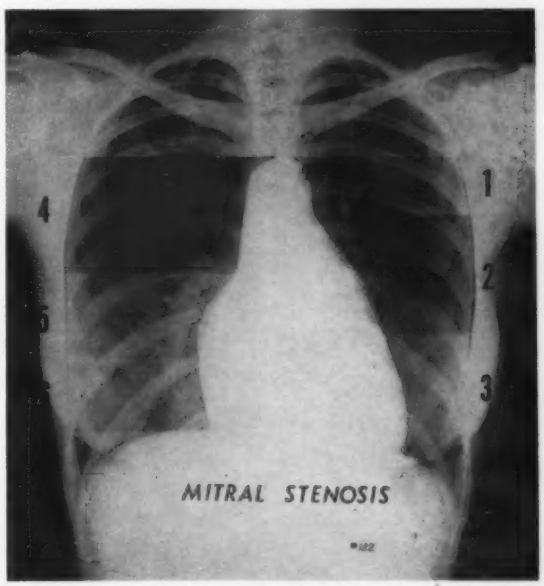


Fig. 5. Typical pattern of mitral stenosis. Note the small aortic knob in Zone 1, the bulge of the left cardiac waistline in Zone 2 and the prominence in Zone 5 due to left atrial enlargement.

P. A. VIEW

SILHOUETTE

Left Border	Zone 1 — Aortic Knob	−	1
	Zone 2 — Waist	+	2
	Zone 3 — Left Ventricular Border		3
Right Border	Zone 4 — Ascending Aorta		4
	Zone 5 — Left Atrium (when visible)	+	5
	Zone 6 — Right Atrium Border		6

DIAGNOSIS *MITRAL STENOSIS*

SILHOUETTE CODE: "+" large, "−" small Add "O" when accentuated

FIG. 6. Coding of mitral stenosis. Significant changes in the zones form a pattern coded as minus 1, plus 2, plus 5.

is the ascending aorta, is normal. Zone 5 is prominent on the basis of the left atrial enlargement, and in this zone we see either a double density or an increased density. Zone 6 is the normal right atrium. In coding mitral stenosis (Fig. 6), therefore, we have a small aorta (coded minus 1), a prominent left cardiac waistline (coded plus 2), and a bulge on the superior aspect of the right atrial border (coded plus 5). Eight typical cases are shown in Figure 7.

Mitral Insufficiency. In mitral insufficiency the mitral valve fails to close completely (Fig. 8). This results in an extreme dilatation of the left atrium secondary to left ventricular backflow. The left ventricle also enlarges. The result of the ensuing back pressure is reflected in the pulmonary vasculature, pulmonary artery-conus segment and right ventricle similar to the hemodynamics found in mitral stenosis.

In the posteroanterior roentgenogram of mitral insufficiency (Fig. 9) we see the changes that occur as anticipated in the schematic drawing (Fig. 8). Zone 1 shows a small aortic knob. Zone 2 reveals a prominent waistline as the result of pulmonary artery-conus enlargement, which is usually more marked in mitral stenosis. Zone 3 represents an enlarged left ventricle. Zone 4 demonstrates a normal ascending aorta. Zone 5 reveals a very markedly enlarged left atrium. Zone 6 shows relative border prominence of the right atrium due to displacement. We therefore note that in coding mitral insufficiency (Fig. 10) we have a small aortic knob (coded minus 1), a prominent left cardiac waistline (coded plus 2), a very prominent left ventricular border (coded plus 3 circled), an extremely large left atrium which overlaps the superior as-

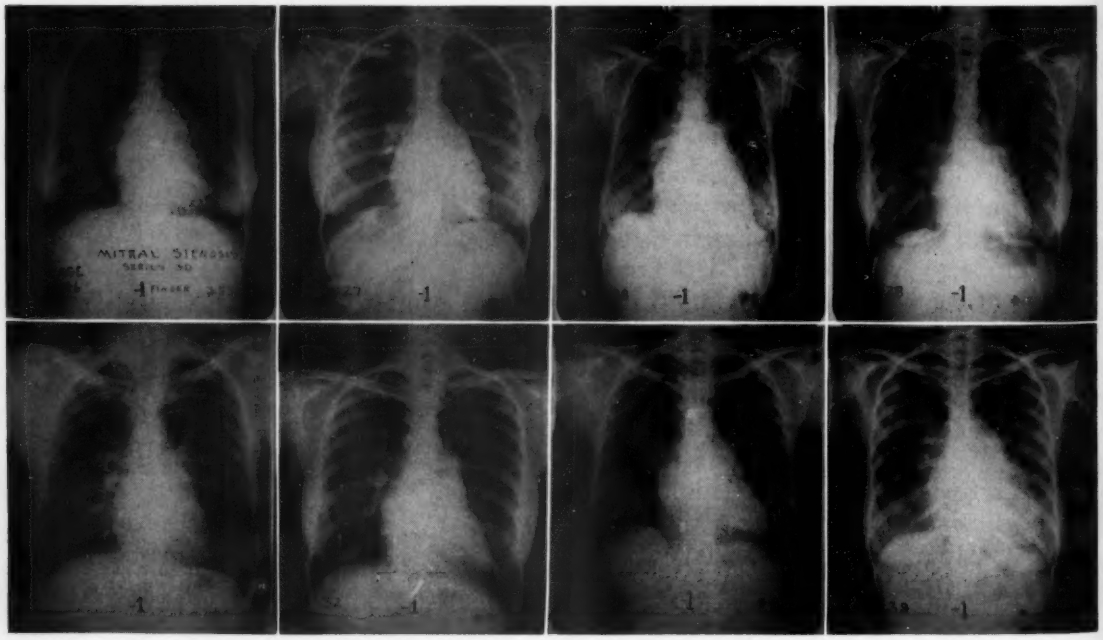
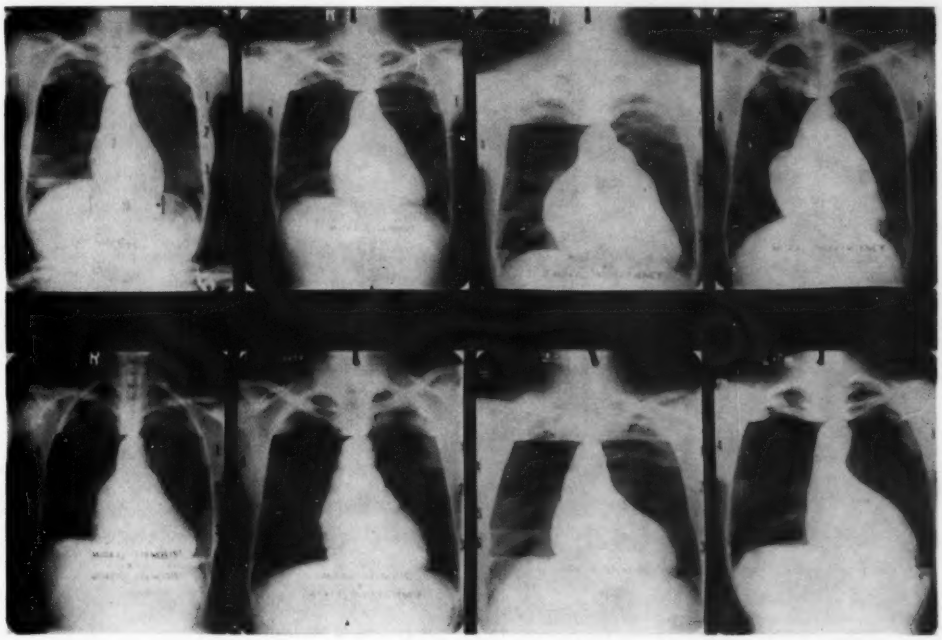


FIG. 7. Eight typical silhouettes of mitral stenosis, showing characteristic changes.



Top Row: Left to right: (1) Normal heart silhouette divided into 6 zones; (2) mitral stenosis—(minus 1, plus 2, plus 5); (3) mitral stenosis and mitral insufficiency—(plus-plus 2, plus-plus 3, plus-plus 5, plus 6); (4) mitral insufficiency—(minus 1, plus 2, plus-plus 3, plus-plus 5, plus 6).

Bottom Row: Left to right: (1) Mitral stenosis and aortic stenosis—(plus 2, plus 3, plus 5); (2) mitral stenosis and aortic insufficiency—(plus 1, plus 3, plus 5); (3) aortic stenosis—(minus 2, plus-plus 3, plus 4); (4) aortic insufficiency—(plus 1, minus-minus 2, plus-plus 3).

pect of the right atrial border (coded plus 5 circled), and a relative enlargement of the right atrium (coded plus 6). Four typical cases are shown in Figure 11.

Mitral Stenosis and Mitral Insufficiency. In this valvular lesion, partial obstruction occurs at the mitral valve due to narrowing of the orifice, and in addition the mitral valve fails to close completely (Fig. 12). The increased work of the left ventricle pumping blood in two directions, backwards into the left atrium and forward into

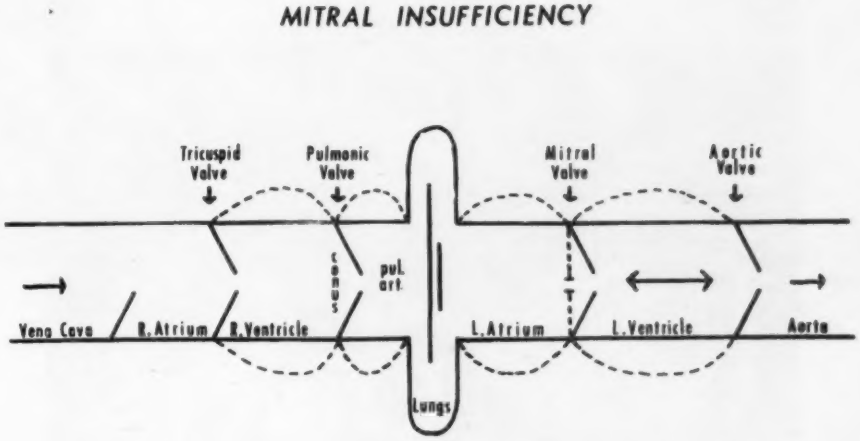


Fig. 8. Schematic drawing of the hemodynamics of mitral insufficiency. Dotted lines indicate enlargements.

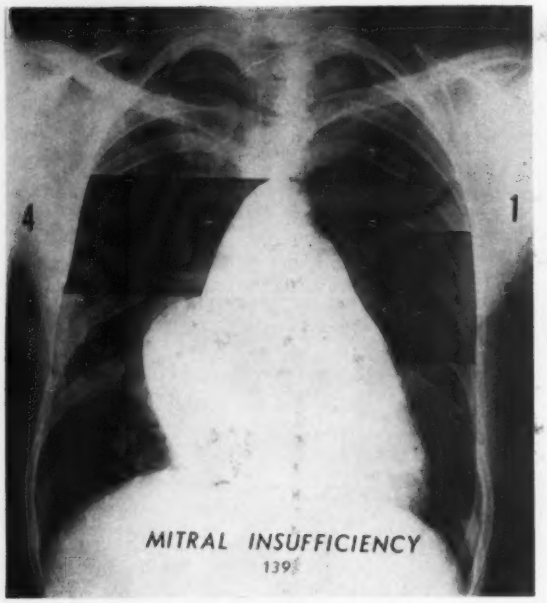


FIG. 9. Mitral insufficiency. Note the prominent left ventricle in Zone 3, the very markedly enlarged left atrium in Zone 5 and general cardiac enlargement.

P. A. VIEW

SILHOUETTE

Left Border	Zone 1 — Aortic Knob	−	1
	Zone 2 — Waist	+	2
	Zone 3 — Left Ventricular Border	⊕	3
Right Border	Zone 4 — Ascending Aorta		4
	Zone 5 — Left Atrium (when visible)	⊕	5
	Zone 6 — Right Atrium Border	+	6

DIAGNOSIS *MITRAL INSUFFICIENCY*

SILHOUETTE CODE "+" large, "−" small. Add "O" when accentuated

FIG. 10. Coding of mitral insufficiency. Significant changes in the zones form a pattern coded as minus 1, plus 2, plus 3 circled, plus 5 circled and plus 6.

the aorta, causes very marked enlargement of the left ventricle. The powerful backflow into the left atrium results in marked enlargement of this cardiac chamber. The result of the ensuing back pressure is reflected in dilatation of the pulmonary vasculature, pulmonary artery-conus segment and right ventricle.

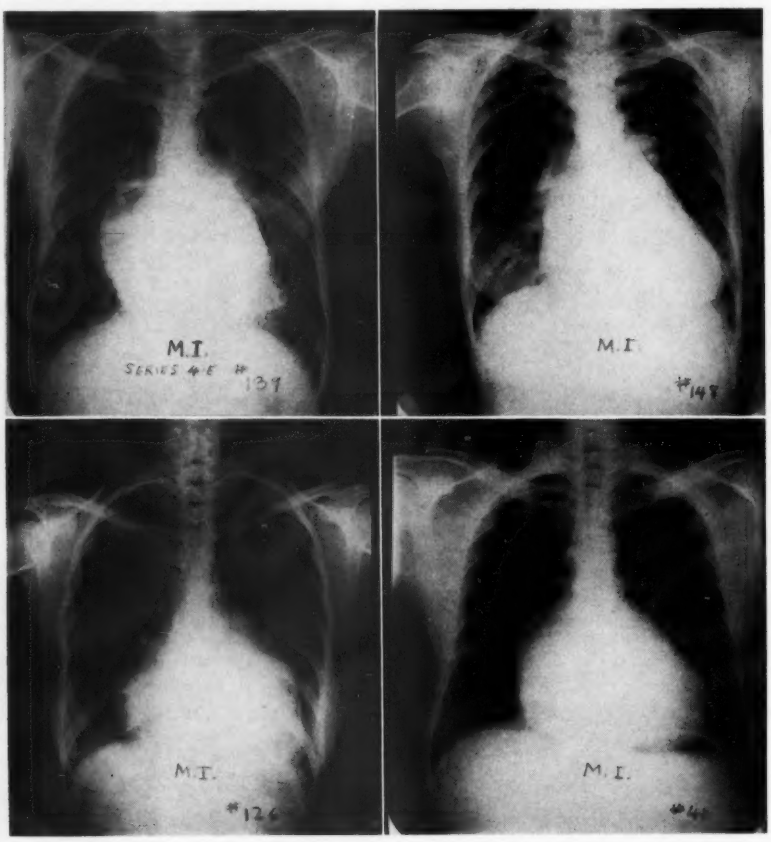


FIG. 11. Four typical cases of mitral insufficiency. In this lesion the left atrium usually becomes extremely large.

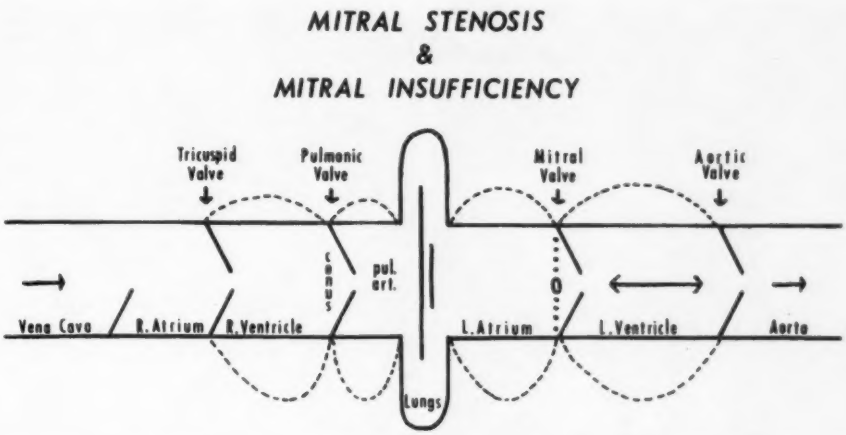


FIG. 12. Schematic drawing of the hemodynamics of mitral stenosis and mitral insufficiency. Dotted lines denote enlargements.

In the posteroanterior roentgenogram of mitral stenosis and mitral insufficiency (Fig. 13), Zone 1 reveals a normal aortic knob. Zone 2 shows a marked prominence of the left cardiac waistline with two distinct bulges. Zone 3 shows a very large left ventricular border. Zone 4 is the normal ascending aorta. Zone 5 shows a very marked enlargement of the left atrium. Zone 6 reveals a relative enlargement of the right atrial border. We therefore note that the typical coded silhouette of mitral stenosis and mitral insufficiency (Fig. 14) presents a double prominence of the left cardiac waistline (coded plus 2 circled), in which the upper bulge denotes the pulmonary artery segment and the lower bulge represents the enlarged pulmonary conus portion of the right ventricle. An associated left ventricular enlargement can also be seen (coded plus 3 circled). Similarly a very marked bulge of the right upper cardiac border (coded plus 5 circled) and a relative right atrial order enlargement (coded plus 6) are observed. Exaggerated individual characteristics of both mitral stenosis and

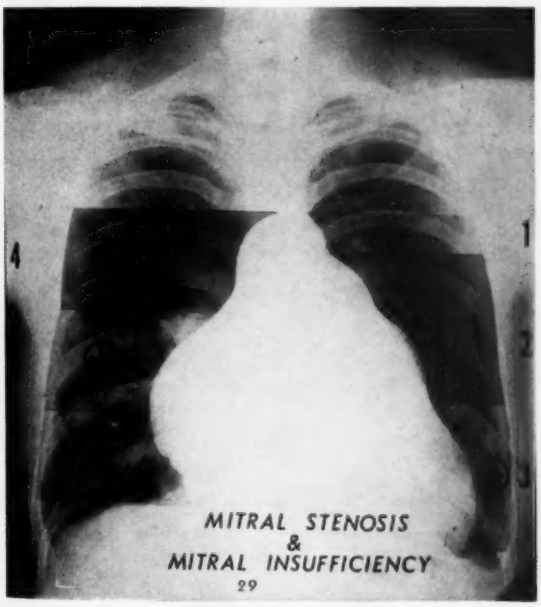


FIG. 13. Mitral stenosis and mitral insufficiency. Note the small aortic knob in Zone 1, the very marked prominence and double contour of the left cardiac waistline in Zone 2, the marked enlargement of the left ventricle in Zone 3 and the marked enlargement of the left atrium in Zone 5. The double contour of the waistline apparently represents the pulmonary artery segment above and the pulmonary conus below.

P. A. VIEW

SILHOUETTE

Left Border	Zone 1 — Aortic Knob		1
	Zone 2 — Waist	⊕	2
	Zone 3 — Left Ventricular Border	⊕	3
Right Border	Zone 4 — Ascending Aorta		4
	Zone 5 — Left Atrium (when visible)	⊕	5
	Zone 6 — Right Atrium Border	+	6

DIAGNOSIS MITRAL STENOSIS & MITRAL INSUFFICIENCY

SILHOUETTE CODE "+" large, "—" small. Add "O" when accentuated

FIG. 14. Coding of mitral stenosis and mitral insufficiency. Significant changes in the zones form a pattern coded as plus 2 circled, plus 3 circled, plus 5 circled and plus 6.

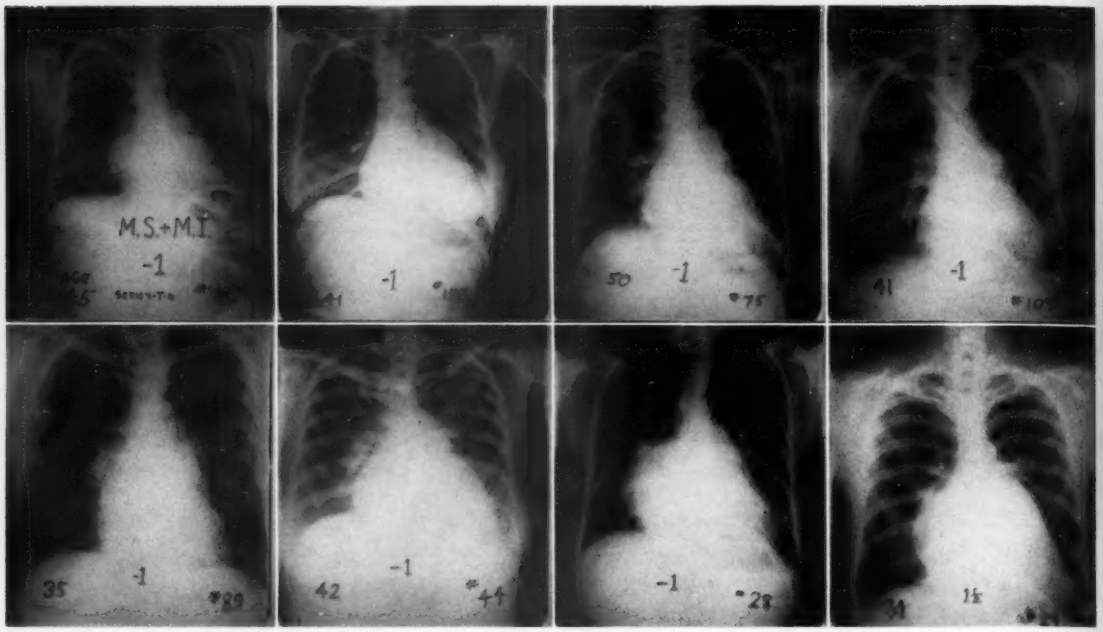


FIG. 15. Eight typical silhouettes of mitral stenosis and mitral insufficiency, showing characteristic changes.

mitral insufficiency are present in this silhouette. The question of which lesion is predominant is extremely important to the surgeon because of the operative techniques. When there is a very marked left atrial enlargement associated with an enlarged left ventricle, it may be presumed that mitral insufficiency is the predominating lesion. Eight typical silhouettes are shown in Figure 15.

Aortic Stenosis. In aortic stenosis partial obstruction occurs at the aortic valve due to narrowing of the orifice (Fig. 16). As a result of this obstruction, the left ventricle enlarges to an extreme degree. This enlargement is self-contained, and it is not reflected backward into the other cardiac chambers or the lungs.

In the posteroanterior roentgenogram (Fig. 17) Zone 1 shows a normal aortic

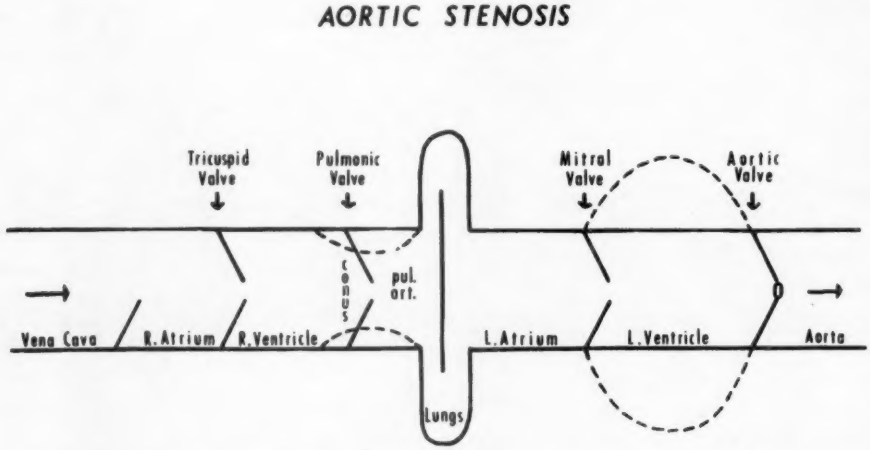


FIG. 16. Schematic drawing of the hemodynamics of aortic stenosis. The outer dotted lines show chamber enlargements. The inner dotted lines in the pulmonic valve sector indicate a deep concavity of the waistline (Zone 2).

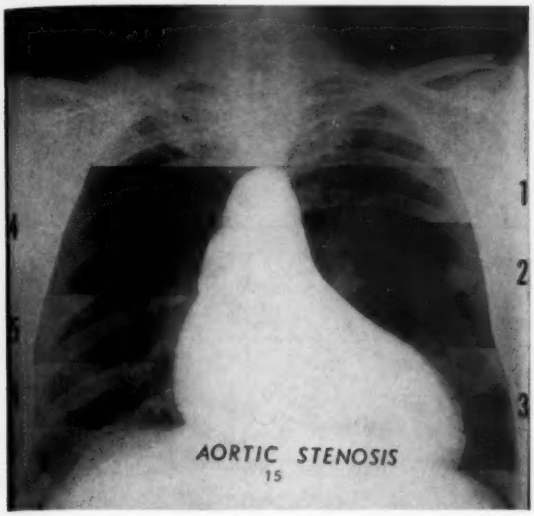


Fig. 17. Aortic stenosis. Note the deep concavity of the left cardiac waistline (Zone 2) uncovering the left hilus, the extreme enlargement of the left ventricle in Zone 3 and the slight accentuation of the ascending aorta in Zone 4.

knob. Zone 2 reveals a deep concavity of the left cardiac waistline, with an uncovered left hilus. Zone 3 shows an extreme enlargement of the left ventricle. Zone 4 reveals a slight accentuation of the ascending aorta. Zones 5 and 6 are normal since no

P. A. VIEW

SILHOUETTE

Left Border	Zone 1 — Aortic Knob		1
	Zone 2 — Waist	−	2
	Zone 3 — Left Ventricular Border	⊕	3
Right Border	Zone 4 — Ascending Aorta	+	4
	Zone 5 — Left Atrium (when visible)		5
	Zone 6 — Right Atrium Border		6

DIAGNOSIS *AORTIC STENOSIS*

SILHOUETTE CODE "+" large, "−" small. Add "O" when accentuated

Fig. 18. Coding of aortic stenosis. Significant changes in the zones form a pattern coded as minus 2, plus 3 circled and plus 4.

atrial enlargements occur. We therefore note that in coding aortic stenosis (Fig. 18), the main changes in the cardiac contours are a deep left waistline (coded minus 2), an extreme enlargement of the left ventricle (coded plus 3 circled) and only slight prominence of the ascending aorta (coded plus 4). Figure 19 shows 8 typical cases of aortic stenosis, the cardiac silhouette being suggestive of a seal balancing a ball on its nose.

Aortic Insufficiency. In aortic insufficiency there is failure of the aortic valve to close completely (Fig. 20). This results in

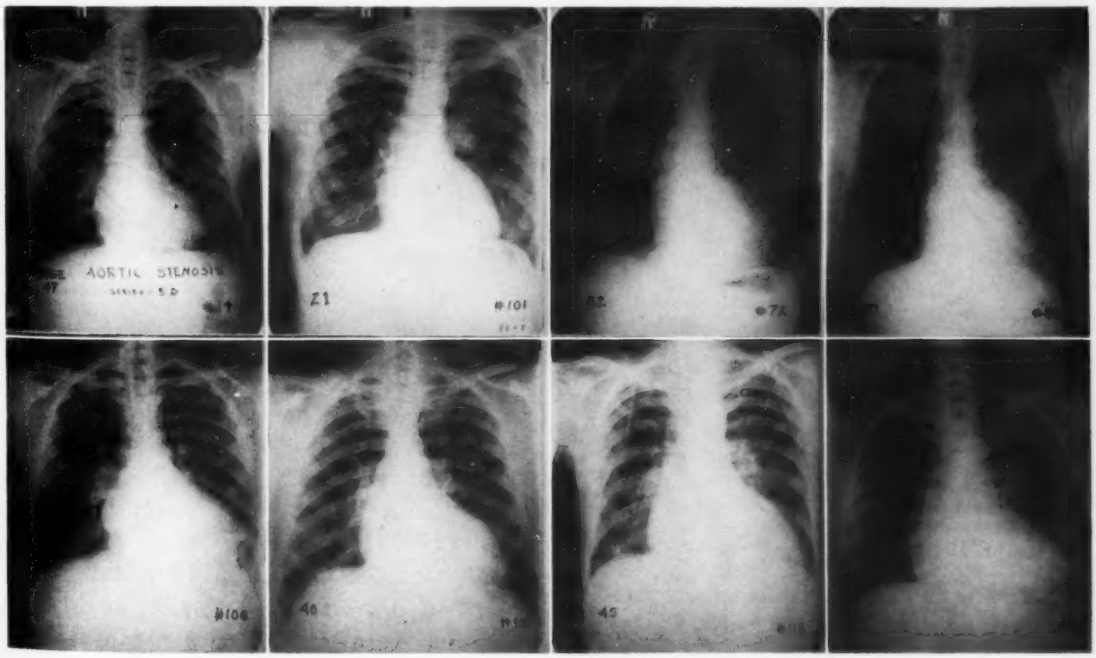


Fig. 19. Eight typical cases of aortic stenosis. The typical silhouette resembles a seal balancing a ball on its nose.

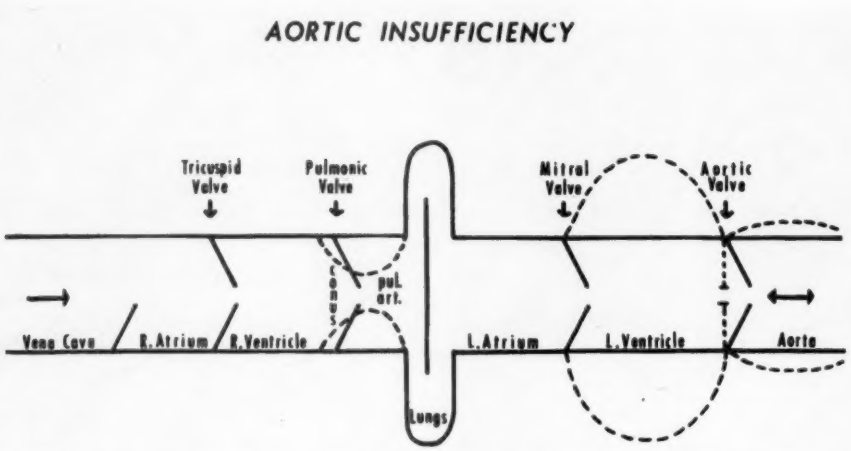


FIG. 20. Schematic drawing of the hemodynamics of aortic insufficiency. Dotted lines demonstrate similar but more extreme changes than in aortic stenosis.

a self-contained enlargement of the left ventricle. There are no other cardiac chamber enlargements.

In the posteroanterior roentgenogram (Fig. 21) Zone 1 shows prominence of the aortic knob. Zone 2 reveals a very deep concavity of the left waistline with uncovering of the left hilus. Zone 3 discloses an extremely large left ventricle. Zone 4 shows

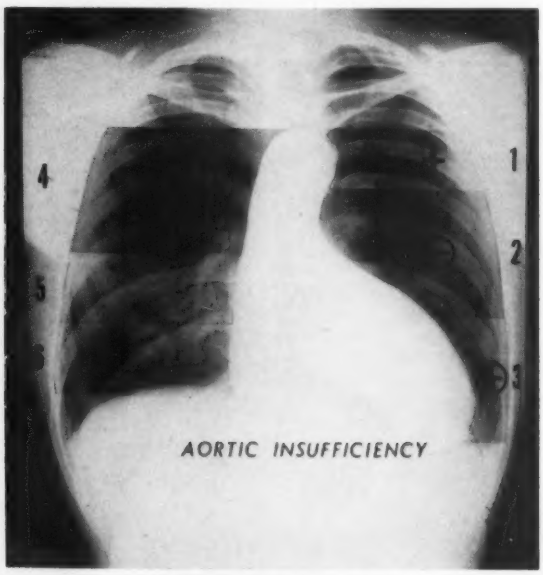


FIG. 21. Aortic insufficiency. Note the prominence of the aortic knob in Zone 1, the deep concavity of the left cardiac waistline in Zone 2 with more uncovering of the left hilus, the extremely large left ventricle in Zone 3 and the normal ascending aorta in Zone 4.

P. A. VIEW

SILHOUETTE

Left Border	Zone 1 — Aortic Knob	+	1
	Zone 2 — Waist	⊖	2
	Zone 3 — Left Ventricular Border	⊕	3
Right Border	Zone 4 — Ascending Aorta		4
	Zone 5 — Left Atrium (when visible)		5
	Zone 6 — Right Atrium Border		6

DIAGNOSIS AORTIC INSUFFICIENCY

SILHOUETTE CODE "+" large, "—" small. Add "O" when accentuated

FIG. 22. Coding of aortic insufficiency. Significant changes in the zones form a pattern coded as plus 1, minus 2 circled and plus 3 circled.

a normal ascending aorta. Zones 5 and 6 are normal, since there is no atrial enlargement. We therefore note that the typical coded silhouette of aortic insufficiency (Fig. 22) presents a prominent aortic knob (coded plus 1), a deep left waistline (coded minus 2 circled) and extreme left ventricular enlargement (coded plus 3 circled). This differs from aortic stenosis mainly by virtue of a prominent aortic knob, a deeper left waistline and more uncovering of the left hilus. This conclusion is based on only one case of pure aortic insufficiency loaned to us by Dr. J. Stauffer Lehman of Hahnemann Medical College and Hospital.

Mitral Stenosis and Aortic Stenosis. In the posteroanterior roentgenogram (Fig. 23). Zone 1 demonstrates a normal aortic

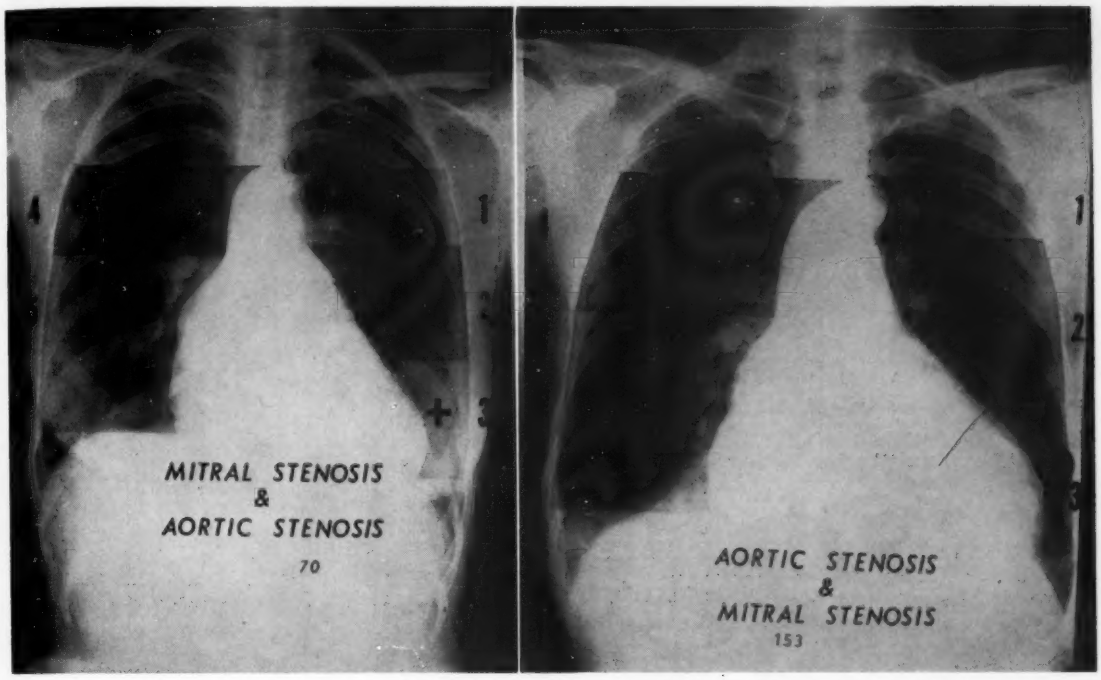


Fig. 23. Mitral stenosis and aortic stenosis. In the roentgenogram on the left where mitral stenosis predominates, the cardiac waistline in Zone 2 is prominent and there is moderate left ventricular enlargement in Zone 3. On the right where aortic stenosis predominates, the waistline is less prominent in Zone 2 and there is more marked left ventricular enlargement in Zone 3. The right cardiac border is similar in both cases.

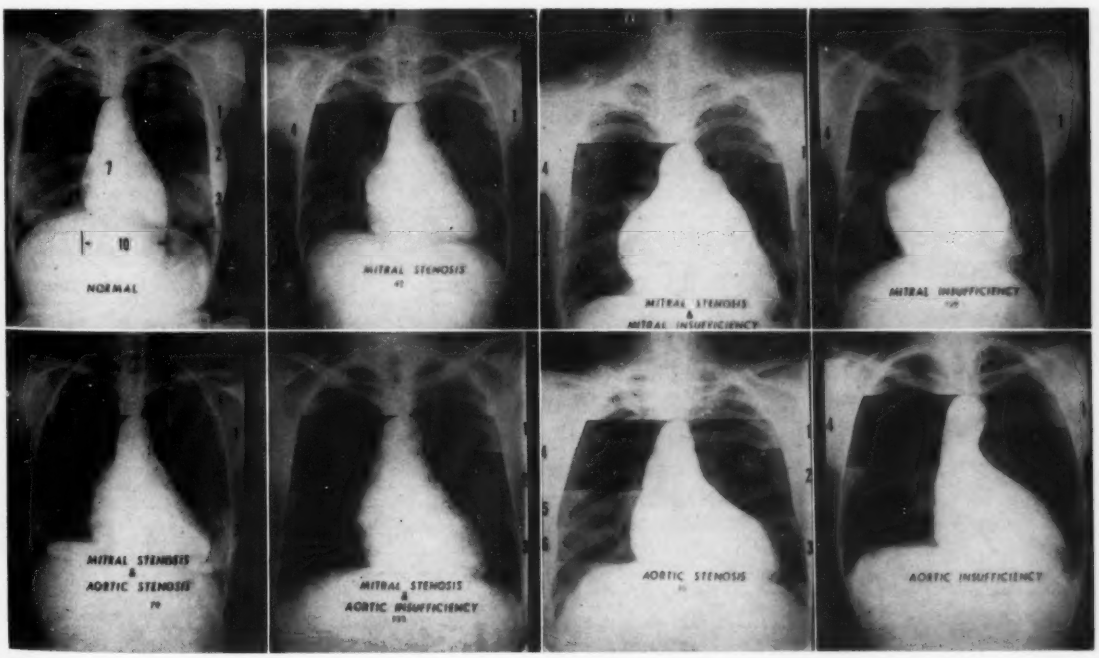


Fig. 24. Posteroanterior view of the normal heart and seven different valvular lesions. Note that the typical silhouettes differ for each valvular lesion.

P.A. VIEW — ZONES — R.H.D.

ZONES	MS	MI	AS	AI	MS MI	MS AS	MS AI	MS AS AI
1	−	−		+			+	+
2	+	+	−	⊖	⊕	+		
3		⊕	⊕	⊕	⊕	+	+	⊕
4			+					
5	+	⊕			⊕	+	+	+
6		+			+			
CASES	94	5	14	1	22	7	1	1

SILHOUETTE CODE "+" LARGE, "−" SMALL. ADD "○" WHEN ACCENTUATED

FIG. 25. Composite coding formula. The individual patterns of the various valvular lesions are combined in one chart. Note that the pattern is different for each valvular lesion.

knob. Zone 2 shows a moderate prominence of the left cardiac waistline. Zone 3 discloses moderate left ventricular enlargement. Zone 4 shows moderate left atrial enlargement. Zone 6 demonstrates a normal right atrium. We therefore note that the typical coded silhouette of mitral stenosis and aortic stenosis presents a moderate enlargement of the left cardiac waistline (coded plus 2), left ventricle (coded plus 3) and left atrium (coded plus 5). This is the usual picture when mitral stenosis predominates in this double lesion. When aortic stenosis predominates, however, the left ventricular border becomes more prominent (coded plus 3 circled), the left cardiac waistline loses its bulge and the right border of the heart remains unchanged.

Composite Typical Silhouettes. A composite of typical cardiac silhouettes (Fig. 24)

Heart Studies -
X-Ray Department – Doctors Hospital

NAME _____

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X-Ray Views	Case No.	Age	Sex	Date	X-Ray No.		
P. A.	LEFT BORDER			Zone 1 — Aortic Knob		1	
				Zone 2 — Waist		2	
				Zone 3 — Left Ventricular Border		3	
	RIGHT BORDER			Zone 4 — Ascending Aorta		4	
				Zone 5 — Left Atrium (when visible)		5	
				Zone 6 — Right Atrium Border		6	
	OTHER CRITERIA			Increased Central Density (Left Atrium)		7	
				Pulmonary Blood Vessels		8	
				Compressed or Elevated Left Bronchus		9	
				Heart Size		10	
	DIAGNOSIS FROM P. A. VIEW						
Lateral	ANTERIOR BORDER			Right Ventricle		11	
R. A. O.	POSTERIOR BORDER			Left Atrium		12	
L. A. O.	ANTERIOR BORDER			Right Auricle (upper)		13	
				Right Ventricle (lower)		14	
	POSTERIOR BORDER			Left Atrium (upper)		15	
				Left Stem Bronchus (Elevated - Compressed)		16	
				Left Ventricle (lower)		17	
	VALVES			Calcifications — (all views)		18	
	FINAL X-RAY DIAGNOSIS						
	OPERATIVE FINDINGS			1. Valve Lesion		19	
				2. Size of Valve Opening		20	
				3. Calcifications		21	
	CLINICAL			1. Diagnosis		22	
				2. Blood pressure		23	
				3. Functional Capacity		24	
	REMARKS						

SILHOUETTE CODE "+" large, "−" small. Add "O" when accentuated _____ M.D.

FIG. 26. Routine work sheet for all views.

consists of posteroanterior studies of various valvular lesions, and it can be noted that the typical silhouette differs for each lesion. These posteroanterior views of the heart are presented in the following order: (1) normal heart; (2) mitral stenosis; (3) mitral stenosis and mitral insufficiency; (4) mitral insufficiency; (5) mitral stenosis and aortic stenosis; (6) mitral stenosis and aortic insufficiency; (7) aortic stenosis; (8) aortic insufficiency.

A composite coding formula (Fig. 25) incorporates into one chart the various codes for all the cardiac lesions studied and it can be noted that the code differs for each lesion.

Other criteria for evaluating cardiac abnormalities that can be observed in the posteroanterior view of these cases are (1) compression or elevation of the left main stem bronchus; (2) dilatation of the major pulmonary blood vessels in the hilar regions; (3) peripheral pulmonary vascular congestion; (4) intracardiac valvular calcifications; and (5) heart size.

CONCLUSION

1. This paper is an attempt to aid in the recognition and differentiation, with a single posteroanterior chest roentgenogram, of various valvular lesions of rheumatic heart disease by the use of a coding formula.

2. This coding formula is dependent on the cardiovascular silhouette in the posteroanterior view, which has been divided into six zones, three on each side. Changes from normal in these zones are recorded by plus or minus symbols.

3. The roentgenographic findings are correlated with the operative findings and form the basis for the development of typical patterns for each lesion described.

4. By applying this method of coding to the cardiovascular silhouette, we hope that these cardiac lesions may be better recognized thereby aiding in the early diagnosis of rheumatic heart disease.

5. It is realized that an insufficient number of cases have been examined, and therefore definite criteria may be revised after future investigations. Only one case of pure aortic insufficiency was available for review.

6. It is hoped that the use of this system of coding of the cardiovascular silhouette can be applied to future studies of all types of cardiac lesions, non-rheumatic as well as rheumatic in origin.

7. Extension of this coding to include all four views of the chest would be an interesting development that could add more criteria for diagnosis.

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We are indebted to Dr. Charles P. Bailey and his associates, Dr. William Jamison and Dr. William Likoff, for the use of the cases presented in this article. Their advice and guidance have proved invaluable. We are particularly grateful to Dr. K. V. S. Rao, of Patna, India, resident in cardiac surgery, who supplied us with most of the operative data.

THE INFLUENCE OF POSTURE ON SIZE AND CONFIGURATION OF THE HEART AS SEEN TELEROENTGENOGRAPHICALLY

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MILWAUKEE, WISCONSIN

A NUMBER of factors may contribute to changes in heart size and configuration as seen roentgenologically when the patient changes from upright to recumbent position or vice versa. These include changes in position of the diaphragm (influencing rotation of the heart on its longitudinal axis), heart rate, flow of blood into the heart, myocardial tonus, and myocardial thickness.[10,11] On assuming the upright position the frontal area as a rule becomes smaller, the reduction at times amounting to 25 per cent or more;[6] whereas, heart volume is said occasionally to decrease by 200 cc. when the patient stands (up to 40 per cent), this degree of change being more common in "small, thin, delicately built people, especially women, the well-developed giant, and many overgrown youths."[13] Likewise, the transverse and usually the broad diameters decrease, while the long diameter may remain the same, decrease, or occasionally increase.[13] Extreme orthostatic reductions are said to occur also in multiple sclerosis, tabes, thyrotoxicosis, emaciation, and in persons with acute cardiac dilatation resulting from toxic and infectious states. The average reduction of heart size is said to be somewhat greater in men than in women.[6]

While considering the upright position indispensable for exact analysis of the cardiovascular complex, the recumbent position is considered best by Zdansky[13] for the most reliable determination of heart size and aortic diameter. He contends that measurements are more constant when made with the patient recumbent than when upright. In contrast, in this country, in medical centers and teaching hospitals, in teleroentgenographic studies made for life insurance companies, in routine fluorophotography during hospital admissions and at induction centers and in large industrial plants, the standing position is quite regularly employed for heart film studies and the standing or sitting position for orthodiagraphy.

True, the hospital radiologist is frequently evaluating the presence or absence of cardiovascular and other intrathoracic diseases by fluoroscopic observations made with the patient in recumbency, as, for example, in the routine barium enema examination and in fluoroscopic studies of the chest and abdomen where the patient's condition does not permit using the sitting or standing posture. Under these conditions the fluoroscopist must keep in mind the position of the patient and its influence on heart size and configuration.

Roesler,[9] discussing cardiac area measurements and variations occurring where area measurements are made by more than one examiner, states that "in the recumbent position the area measurements are on the average 20% greater."

Concerning the so-called hypoplastic or "small" heart, Zdansky[13] contends that these hearts are frequently actually not smaller than normal but only appear so with the subject standing, because in this position they are inadequately filled with blood. With deficient muscle tonus of the diaphragm and abdominal muscles and also perhaps because of vasomotor weakness, a portion of the blood is retained in the splanchnic area with the patient upright, but when assuming recumbency this blood, amounting to as much as 1.5 liters, is brought back into circulation and immediately the heart takes on a "normal" appearance.

On the other hand, Hodges,[2] discussing the usefulness of various positions for car-

diac orthodiascopy, states, "The standing or sitting frontal projections were more dependably reproducible on repeated occasions for the same individual than when any other posture was used. Furthermore, the free area of cardiac silhouette above the diaphragm was proportionately greater under these circumstances."

Various workers—physiologists, cardiologists,[11] radiologists—have on one occasion or another referred to changes in heart size occurring with changes in posture of the patient but have given little indication of what may ordinarily be expected or of the frequency with which an important change in size occurs.

Discussing specifically the factors of greatest importance in influencing change in heart size with change in body position, Kjellberg[4] and Larsson[5] state that with the patient in the lying position the heart volume undergoes no demonstrable change with pulse increases up to 120 per minute, but, on the other hand, in the upright position a diminution in heart volume proportional to the elevated pulse rate takes place. The pulse rate must rise above 75 to 80 per minute before any volume decrease can be observed. This observation would indicate that a person with a lying pulse of, for example, 66 and a standing pulse of 78 might show no change in heart volume. This is in agreement with our findings that very frequently the change in the frontal area measurement is so small as to be negligible. The difference in heart volume between lying and standing positions is said not to be proportional to the difference between the pulse rate in the respective positions, but to the standing pulse rate. Kjellberg and Larsson were not able to confirm large volume differences up to 40 per cent between lying and upright positions found by Nylin.[7] Kjellberg[4] has contended that cases of early or mild mitral valvular disease were not diagnosed correctly roentgenologically because the patient was examined in sitting or standing positions. Under these circumstances, he maintains, the heart chambers are insufficiently filled to show compression and/or displacement of the barium-filled esophagus. Kjellberg, therefore, recommends the recumbent position, preferably prone. Ideally the roentgenographic exposure for the recumbent esophagram should be done at the end of systole at the termination of the T-wave in the electrocardiogram. Schorr and co-workers[10] at Hadassah University Hospital in Jerusalem confirmed Kjellberg's observations and indicated that in their series there were no "false positives" in normal patients.

The opposite viewpoint concerning the preferred position for the detection of organic left auricular englargement as occurs in mitral valvular disease is held by Kaye, Meyer, Van Lingen, McGregor, and Vraudo[3] who reported the roentgenologic findings in 100 cases of clinically accepted mitral valvular disease and 100 control patients and found that, "in many normal cases when radiographed in the recumbent position, backward displacement of the barium-filled esophagus is noted," and in the recumbent position they were not able to assess the degree of displacement which should be considered of pathologic significance. The observation of these workers is in agreement with the present author who found a 27 per cent incidence of some degree of posterior displacement, either characteristic or somewhat atypical, at the level of the left auricle in normal individuals when the patient was examined in recumbent position. In a series of persons[4] of whom esophagrams were made both in upright and recumbent positions, the incidence of "false positive" posterior displacement of the barium-filled esophagus, occurring when only the upright position was used for evaluation, was 10 per cent as determined by Kaye and his co-workers at Johannesburg Hospital.

TECHNIQUE OF STUDY

In the beginning of the study a few patients were examined by conventional posteroanterior standing teleroentgenogram and prone (posteroanterior recumbent)

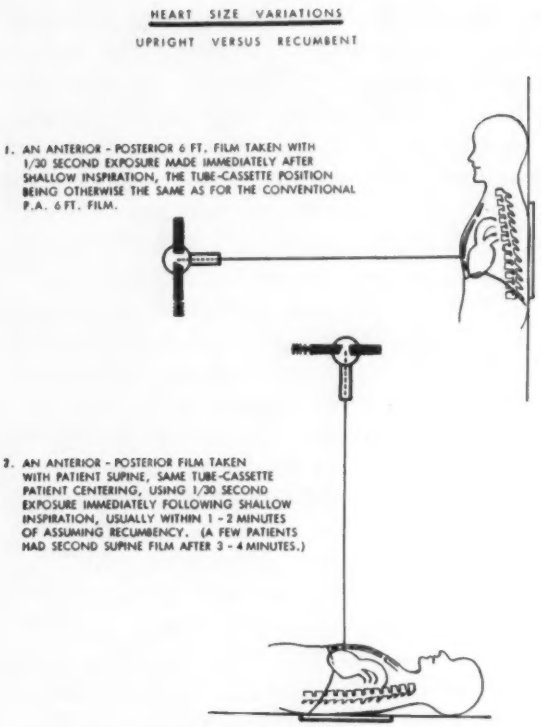
HEART SIZE VARIATIONS
UPRIGHT VERSUS RECUMBENT
1. AN ANTERIOR - POSTERIOR 6 FT. FILM TAKEN WITH 1/30 SECOND EXPOSURE MADE IMMEDIATELY AFTER SHALLOW INSPIRATION, THE TUBE-CASSETTE POSITION BEING OTHERWISE THE SAME AS FOR THE CONVENTIONAL P.A. 6 FT. FILM.
2. AN ANTERIOR - POSTERIOR FILM TAKEN WITH PATIENT SUPINE, SAME TUBE-CASSETTE PATIENT CENTERING, USING 1/30 SECOND EXPOSURE IMMEDIATELY FOLLOWING SHALLOW INSPIRATION, USUALLY WITHIN 1 - 2 MINUTES OF ASSUMING RECUMBENCY. (A FEW PATIENTS HAD SECOND SUPINE FILM AFTER 3 - 4 MINUTES.)

FIG. 1. Each patient was examined by standing anteroposterior and recumbent (supine) teleroentgenograms, and the transverse diameters and frontal areas were compared.

teleroentgenogram, each taken at the end of normal shallow inspiration, and each within one to two minutes of the patient being placed in position against the cassette. In a small number of cases the patient was kept recumbent another three to five minutes and a second recumbent teleroentgenogram exposed. In none of these few "prolonged recumbency" teleroentgenograms was there appreciable change in the heart measurements as compared with those obtained from the standard technique recumbent position teleroentgenograms. It was soon found that the prone position was not an easy one for some patients to assume or maintain; hence, the positions thereafter employed routinely were the anteroposterior standing and supine (anteroposterior recumbent). These latter positions proved easier for the patients to assume (they were all ambulatory), and it was furthermore considered preferable to use supine rather than prone in noting the influence of change of position on heart size, since bedside heart studies are frequently done with the patient supine, and nearly all roentgenoscopic evaluations of cardiac disease done during barium enema and some other fluoroscopic studies of hospital patients are likewise done in supination (Fig. 1). (Zdansky[13] has stated a preference for the supine position in evaluating heart size, while Kjellberg[4,5] prefers to make evaluations with the patient prone.) Care was exercised to keep the point of centering over the upper sternum, and, whenever it appeared that there was an appreciable difference in position of the diaphragm on the two teleroentgenograms, the study was repeated. From these paired films, transverse diameter and frontal area determinations were obtained and the increase or decrease of these measurements recorded (Tables I and II). The changes were expressed also in percentage increase or decrease. From an experience of twenty-five years of practice doing serial orthodiagraphic and teleroentgenographic studies of ambulatory cardiac patients, it was considered that percentage changes of less than 6 or 7 per cent would not ordinarily be considered indicative of frank changes in heart size, since such variations are probably within the limits of error of roentgen cardiac measurement.

In the present series of 70 patients who had upright and recumbent teleroentgenogram comparisons, and also in a previous series of about 100 normal and abnormal cardiac patients esophagrams also were done with the patient in upright and recumbent right anterior oblique positions (Fig. 2). In the series of 70 patients herein reported, attention was paid to possible correlation of changes in heart size as noted on the frontal teleroentgenograms and changes in the course of the barium-filled esophagus. While frequently but not constantly (Fig. 8) we did observe more displacement and/or compression of the barium-filled esophagus in recumbency in the patients with organic mitral disease,

TABLE I

25 CLINICALLY AND ROENTGENOLOGICALLY NORMAL HEARTS
COMPARISON OF UPRIGHT AND RECUMBENT TELEROENTGENOGRAM TRANSVERSE DIAMETERS AND FRONTAL AREAS

Case No.	Patient	Sex	Age	Height (inches)	Weight (pounds)	Clin. Classif.	Cardiac T.D.		Per Cent Change	Cardiac Area		Per Cent Change
							Vert.	Horiz.		Vert.	Horiz.	
1	J.L.	M	34	68	176	normal	15.3	15.8	+3.	148	157	+6.
2	M.S.	M	15	70	140	normal (funct. syst. murmur)	14.1	15.0	+6.3	139	131	−5.6
3	J.K.	F	37	62	123	normal	13.7	13.4	−2.4	114	108	−5.2
4	R.P.	M	28	67	190	normal	14.9	14.7	−1.4	117	122	+4.2
5	H.M.	M	44	72	195	normal	13.3	13.4	+0.7	134	136	+1.6
6	E.R.	M	62	68	152	normal	12.5	12.4	−0.8	124	126	+1.2
7	M.O.	F	33	63	133	normal (funct. syst. murmur)	11.4	12.4	+9.0	107	106	−0.8
8	C.B.	F	37	66	150	normal	14.2	13.2	−7.0	131	128	−2.3
9	W.M.	M	31	70	167	normal (funct. syst. murmur)	12.8	14.3	+12.0	113	134	+18.8
10	R.D.	M	25	69	140	normal	11.7	13.7	+17.0	110	137	+24.5
11	R.G.	M	40	74	175	normal (funct. syst. murmur)	11.7	13.3	+13.8	109	117	+7.2
12	R.B.	M	32	75	200	normal	13.2	13.7	+4.0	131	136	+4.0
13	A.B.	M	56	74	193	normal	14.6	14.9	+2.0	151	143	−5.3
14	A.C.	M	51	68	161	normal	11.9	12.2	+2.8	93	92	−1.0
15	F.K.	M	46	72	175	normal	14.2	14.0	−1.4	143	132	−7.6
16	R.S.	M	24	69	128	normal (iatrogenic heart disease, funct. murmur)	12.3	13.4	+9.0	118	136	+15.2
17	J.S.	F	63	66	134	normal (pseudo-angina)	14.6	14.5	−0.6	137	132	−3.6
18	M.S.	F	28	62	220	normal	13.9	13.4	−3.5	109	109	0
19	W.S.	M	23	72	168	normal	12.8	14.7	+15.0	115	141	+22.6
20	H.T.	F	37	66	161	normal (iatrogenic heart disease, sternal depress)	15.2	14.8	−2.6	155	142	−8.2
21	E.M.	M	54	69	170	normal	15.3	15.7	+2.7	134	139	+4.0
22	L.F.	F	29	69	145	normal	13.6	14.3	+5.0	121	131	+8.3
23	E.G.	M	40	75	180	normal (iatrogenic heart disease, sternal depress)	13.8	15.9	+15.2	142	149	+5.0
24	M.Z.	F	32	64	125	normal (post-thyroid)	12.4	12.6	+1.7	117	117	0
25	R.O.	M	14	68	138	normal	12.9	13.9	+8.0	128	143	+11.7

The greatest increase in cardiac transverse diameter was 17%, the largest decrease 7%; average, increase of 4%.
The greatest increase in frontal area was 24%, the largest decrease 8%; average, increase of 4%.

we did not, as indicated previously, regularly record significant increases in frontal measurements, particularly the area one, in this same group (Fig. 8, *A* and *B*). Furthermore, cases which showed some of the largest changes in heart size in recumbency as compared to the upright position, showed no changes in the course of the barium-filled esophagus (Fig. 10, *C* and *D*). But a rather large number of patients, in whom there was no clinical evidence of mitral valvular disease or other disease which might cause left auricular enlargement, did show changes in the course of the barium-filled esophagus when examined in the recumbent position (Fig. 6 and 7). The incidence of "false positive" esophagrams was 27 per cent in recumbency, a much larger incidence than the 10 per cent "false positive" incidence noted by Kaye, Meyer, Van Lingen, McGregor, and Vraudo in their analyses of 100 normal and 100 mitral valvular disease patients when relying on the upright position for esophagram evaluation. Because of this experience we agree with Kaye and coworkers that the patient should be studied in the upright position when looking for esophagram evidence of left auricular enlargement.

TABLE II

45 CLINICALLY ABNORMAL HEARTS

Case No.	Patient	Sex	Age	Height (inches)	Weight (pounds)	Clin. Classif.	Cardiac T.D.		Per Cent Change	Cardiac Area		Per Cent Change
							Vert.	Horiz.		Vert.	Horiz.	
						Group I: Valvular Disease						
1	A.P.	F	61	64	114	mitral and aortic regurgitation (episodes of heart failure)	14.9	15.2	+2.0	171	172	+0.5
2	W.H.	M	72	70	152	aortic stenosis and insufficiency	15.4	15.4	0	138	141	+2.2
3	C.U.	F	66	62	122	mitral regurg. and aortic stenosis	11.6	12.5	+7.8	97	99	+2.0
4	G.D.	F	50	62	137	aortic insuffic.	14.9	14.8	−0.5	132	131	−0.7
5	D.J.	M	38	67	168	aortic stenosis and regurgitation	19.0	19.1	+0.6	188	193	+2.6
6	J.L.	M	57	69	130	mitral disease	13.7	14.7	+7.3	156	157	+0.7
7	E.G.	F	39	66	138	mitral disease	13.9	13.7	−1.3	133	125	−6.0
8	J.B.	M	28	64	170	aortic insuffic.	15.3	15.3	0	137	140	+2.1
9	J.A.	M	49	67	145	valvular aortic and possibly mitral disease	17.0	17.7	+4.0	185	191	+3.2
10	D.R.	M	28	72	175	mitral stenosis, calcified	18.3	18.2	−0.5	186	184	−1.2
11	H.S.	F	30	62	118	mitral disease, stenosis predominating, 10 mo. postoperative	15.5	14.8	−4.4	148	143	−3.3
12	R.M.	M	41	72	150	mitral regurg. with auricular fibrillation	16.8	16.9	+0.7	235	234	−0.5
13	J.K.	F	19	63	126	early mitral stenosis	11.0	11.9	+8.2	103	116	+12.6
14	A.S.	F	65	61	120	mitral stenosis with episodes of heart failure	13.9	13.4	−3.6	108	114	+5.6
15	J.W.	F	25	62	123	mitral stenosis and regurgitation	16.3	16.8	+3.0	174	186	+7.0
						Group II: Arteriosclerotic Heart Disease With or Without Infarction						
1	J.F.	M	45	71	200	coronary artery disease, minimal electrocardiographic changes	15.0	14.9	−0.7	130	133	+2.2
2	H.H.	M	61	69	200	coronary disease	15.6	15.5	−0.6	143	145	+1.5
3	P.S.	M	62	64	156	old coronary occlusion	16.6	16.8	+1.1	158	164	+4.0
4	C.M.	M	66	68	137	old myocardial infarction	14.0	14.1	+0.8	130	132	+1.4
5	R.H.	M	82	66	167	coronary sclerosis, minimal electrocardiographic changes	15.9	15.5	−2.4	136	133	−2.0
6	B.R.	M	63	68	168	arteriosclerotic heart disease, recent failure	15.3	15.7	+2.7	147	149	+1.2
7	W.H.	M	53	72	177	old coronary thrombosis	16.7	15.3	−8.3	136	137	+0.8
8	G.G.	M	53	67	140	old coronary thrombosis	12.2	12.6	+3.3	96	103	+7.3
9	R.F.	M	60	67	154	old myocardial infarction	16.7	16.0	−4.2	155	158	+2.0
10	J.O.	M	59	68	192	coronary artery disease with angina	16.6	16.4	−1.0	161	163	+1.2
11	R.B.	F	44	66	173	coronary artery disease, angina, atypical	10.9	11.4	+4.6	88	93	+5.7
12	C.W.	M	68	74	210	arteriosclerotic heart disease with mitral regurg.	14.4	13.7	−5.0	125	123	−1.5
						Group III: Hypertensive Heart Disease						
1	T.S.	M	49	72	265	hypertension, moderate	16.0	17.0	+6.1	150	155	+3.3
2	M.R.	F	72	64	150	mild hypertensive disease with arteriosclerosis	14.3	14.7	+3.0	130	132	+1.5
3	V.K.	M	58	66	183	moderate hypertensive heart disease	15.2	16.0	+5.2	142	151	+6.2
4	F.W.	F	60	72	160	hypertensive heart disease, long-standing	14.4	14.9	+3.4	136	140	+3.0
5	W.H.	M	57	68	163	moderately severe hypertensive heart disease	15.0	15.0	0	135	134	−0.7
						Group IV: Congenital Heart Disease						
1	M.L.	M	6	49	54	pulmonary valvular stenosis, 2 yr. postoperative	13.7	14.2	+3.7	123	126	+2.2
2	J.H.	M	4	42	39	probable pulmonary stenosis	8.4	8.6	+2.2	54	58	+7.4

Table II—*Continued*

Case No.	Patient	Sex	Age	Height (inches)	Weight (pounds)	Clin. Classif.	Cardiac T.D.		Per Cent Change	Cardiac Area		Per Cent Change
							Vert.	Horiz.		Vert.	Horiz.	
3	R.B.	M	16	70	170	patent interventricular septum	13.2	14.3	+8.3	130	133	+2.3
4	A.H.	F	38	67	158	patent interventricular septum	13.2	14.2	+7.8	124	131	+5.7
5	G.F.	M	27	75	180	congenital heart, probable interventricular septal defect	15.7	15.9	+1.2	164	174	+6.0
6	S.K.	F	4	41	40	congenital heart, probable patent interventricular septal defect	9.6	10.7	+11.5	62	69	+11.3
7	C.C.	F	5	48	46	congenital heart, patent ductus	10.4	10.5	+1.0	68	69	+1.4
8	D.P.	F	12	62	104	probable congenital heart disease, ?interventricular septal defect	11.1	12.6	+13.4	81	96	+18.5
						Group V: Miscellaneous						
1	S.B.	M	46	67	178	recurring episodes heart failure, deficiency state? (beriberi)	16.0	18.1	+13.1 (*1952*)	156	182	+16.6
							19.8	20.3	+2.5 (*1953*)	214	217	+1.3
2	A.W.	M	37	69	158	recent pericarditis	13.4	14.2	+6.0	121	124	+2.5
3	A.L.	M	56	69	140	myocardial degeneration, etiology undetermined	16.4	16.3	−0.6	199	195	−2.0
4	W.B.	F	62	67	125	auricular fibrillation, ventricular septal defect?	11.2	11.4	+1.9	95	101	+6.3
5	E.P.	M	62	68	206	peripheral vascular disease	14.6	14.4	−1.4	133	130	−2.3

The greatest increase in cardiac transverse diameter was 13%, the largest decrease 8%; the average change, an increase of 2% for the 45 abnormal hearts.

The greatest increase in frontal area was 18%, the largest decrease 6%; the average change, an increase of 3% for the 45 abnormal hearts.

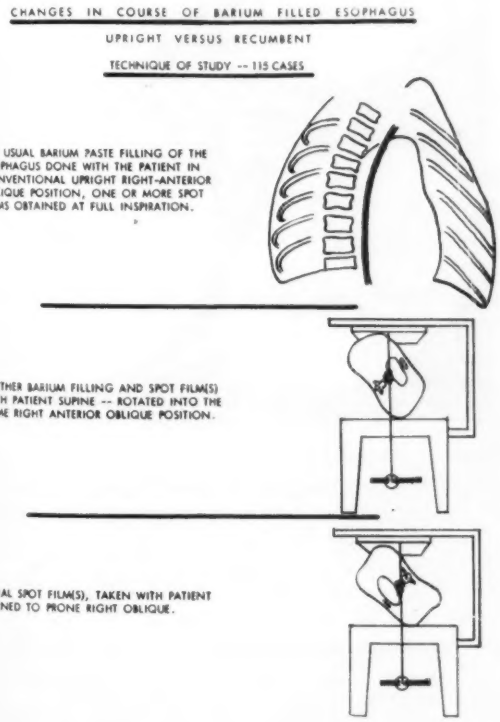
CHANGES IN COURSE OF BARIUM FILLED ESOPHAGUS
UPRIGHT VERSUS RECUMBENT
TECHNIQUE OF STUDY -- 115 CASES
1. THE USUAL BARIUM PASTE FILLING OF THE ESOPHAGUS DONE WITH THE PATIENT IN CONVENTIONAL UPRIGHT RIGHT-ANTERIOR OBLIQUE POSITION, ONE OR MORE SPOT FILMS OBTAINED AT FULL INSPIRATION.
2. FURTHER BARIUM FILLING AND SPOT FILM(S) WITH PATIENT SUPINE -- ROTATED INTO THE SAME RIGHT ANTERIOR OBLIQUE POSITION.
3. FINAL SPOT FILM(S), TAKEN WITH PATIENT TURNED TO PRONE RIGHT OBLIQUE.

Fig. 2. This group, and another group studied previously, had esophagrams in the usual upright right anterior oblique and also in recumbent, prone, and supine oblique positions, and the findings were compared.

FINDINGS

Almost from the beginning we were impressed with the *absence* of regularly occurring significant increase in heart size when the patient assumed the recumbent position (Tables I and II, Fig. 3). In a few cases in both the normal and abnormal heart groups there were *decreases* of 6 to 8 per cent in transverse diameter and/or area when the patient was placed recumbent. *Increases* in both transverse diameter and area were, of course, much more common. Using a thin-walled rubber balloon, roughly approximating the form of the human heart in frontal projection, and beginning with the model filled with

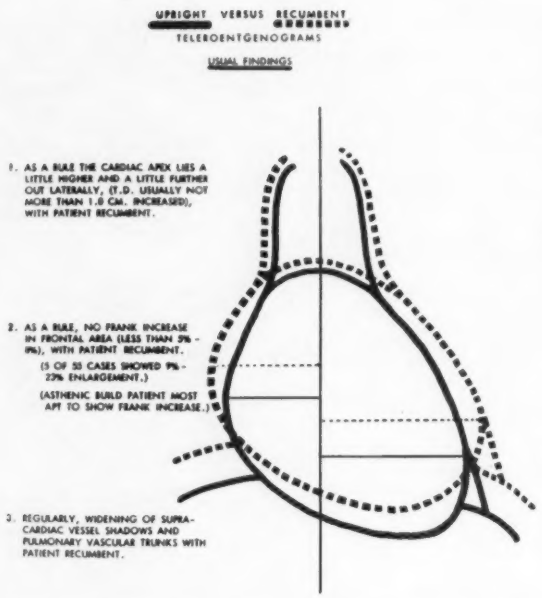
UPRIGHT VERSUS RECUMBENT
TELEROENTGENOGRAMS
USUAL FINDINGS
1. AS A RULE THE CARDIAC APEX LIES A LITTLE HIGHER AND A LITTLE FURTHER OUT LATERALLY, (T.D. USUALLY NOT MORE THAN 1.0 CM. INCREASED), WITH PATIENT RECUMBENT.
2. AS A RULE, NO FRANK INCREASE IN FRONTAL AREA (LESS THAN 5% - 9%), WITH PATIENT RECUMBENT.
(5 OF 50 CASES SHOWED 9% - 23% ENLARGEMENT.)
(ASTHENIC BUILD PATIENT MOST APT TO SHOW FRANK INCREASE.)
3. REGULARLY, WIDENING OF SUPRA-CARDIAC VESSEL SHADOWS AND PULMONARY VASCULAR TRUNKS WITH PATIENT RECUMBENT.

Fig. 3. Composite frontal silhouette from upright and supine teleroentgenograms. The "physiologic dilatation" was only occasionally marked, the average increase in transverse diameter and area was less than 5 per cent.

400 cc. water, teleroentgenogram measurements of "transverse diameter" and "frontal area" of the water-filled model were made with the balloon distended to 400, 500, 600, and 700 cc. volumes (Fig. 4). The model was placed on a block approximately 8 cm. above the cassette in an effort to approximate the relationships of the model and cassette to those of the *in vivo* hearts with the patient in recumbency. It is noted in this experimental study that 10 to 16 per cent increases of "frontal area" occurred with each increase of 100 cc. fluid content. Transverse diameter increases ranged from 4 to 9 per cent. It would seem probable that equal increases in blood content in the functioning heart would produce greater degrees of cardiac enlargement as measured roentgenographically because of the thickness of the heart walls and the presence of interauricular and interventricular septa and valves which together contribute a considerable part of the total heart volume.

No attempt was made in this study to measure simultaneously changes in the lateral cardiac silhouette—either linear or area. A study by electrokymography of changes in posteroanterior cardiac diameter was done recently by Eddleman, *et al.*[1] They found the posteroanterior cardiac diameter *smaller* in 9 out of 10 normal young male medical students when the patient was supine as compared to the upright teleroentgenogram diameter. The authors believed the smaller posteroanterior cardiac diameter with the subject recumbent was attributable to a change in heart *form* and did not indicate a true volume decrease. Such a change in form may conceivably occur, producing an increase in cardiac volume beyond that which our frontal teleroentgenogram measurements would permit one to expect.

In the series of 25 clinically and roentgenologically normal hearts the greatest increase in *transverse diameter* was 17 per cent (from 11.7 cm. to 13.7 cm.), while the

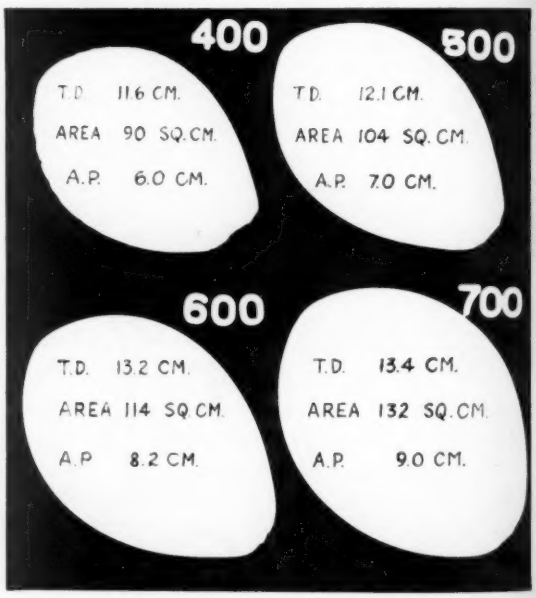

Fig. 4. Teleroentgenograms of a heart model mounted 8 cm. above cassette to simulate the anteroposterior teleroentgenogram supine studies of the normal and abnormal hearts. Comparison of "transverse diameters," "frontal areas," and anteroposterior diameters of water-filled balloon containing 400 cc., 500 cc., 600 cc., and 700 cc. Volume increases of 100 cc. caused frontal area increases of 10 to 16 per cent, while the transverse diameters increased 4 to 9 per cent.

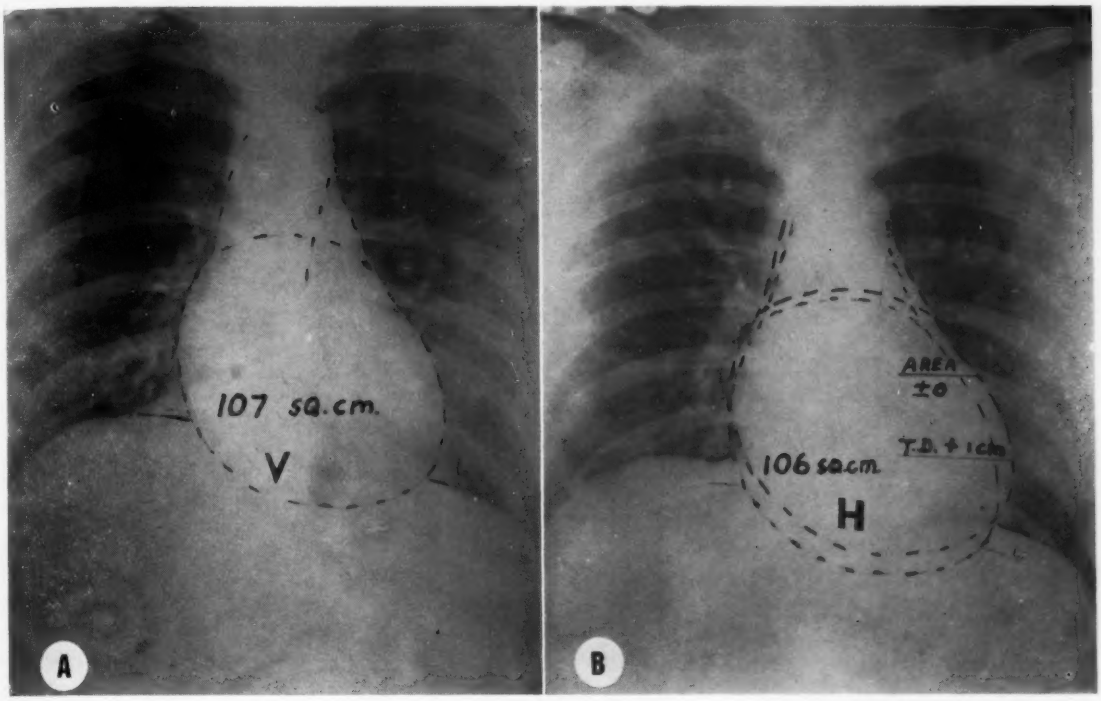

FIG. 5. M.O. Normal heart. Female, aged thirty-three. Height 63 inches, weight 133 pounds. Pulse standing 90, recumbent 76. (*A*) Upright teleroentgenogram. (*B*) Supine teleroentgenogram. Upright film cardiovascular contour superimposed on recumbent film cardiovascular outline. Transverse diameter increased 1 cm. with patient recumbent; frontal area showed no significant change.

greatest decrease was 7 per cent (from 14.2 cm. to 13.2 cm.). The average change was a negligible 4.3 per cent increase (roughly 0.5 cm.) and the median deviation 2.8 per cent increase when the supine teleroentgenogram measurement was compared to the upright.

In the same group of normal hearts the largest increase in frontal *area* was 24.1 per cent (from 110 sq. cm. to 137 sq. cm.), and the greatest decrease was 8 per cent (from 143 sq. cm. to 132 sq. cm.). The average change was an increase of 3.8 per cent (approximately 5 sq. cm.), while the median change was 1.6 per cent (Fig. 6 and 7).

The changes noted in the group of 25 normal hearts were not very different from the changes noted in the abnormal heart groups consisting of 45 patients (2 of whom had repeat studies after a lapse of time).

In the abnormal series the greatest increase in *transverse diameter* was 13 per cent (from 11.1 cm. to 12.6 cm.), the largest decrease 8 per cent (from 16.7 cm. to 15.3 cm.) with the average change in transverse diameter being an increase of 2.3 per cent and the median deviation 1.9 per cent.

Frontal *area* changes in the abnormal series showed a maximum increase of 18 per cent (from 81 sq. cm. to 96 sq. cm., a twelve year old female patient with a probable congenital lesion, unclassified). The maximum decrease was 6 per cent (from 133 sq. cm. to 125 sq. cm., a patient with rheumatic mitral valve disease). The average change was an increase of 3.2 per cent and the median change an increase of 2.2 per cent.

That changes in functional capacity of a diseased heart may influence importantly the degree of change in heart size occurring with change of position is indicated by measurements made on Case No. 1 of the Miscellaneous Disease Group (Fig. 9).

This patient was not positively classified as to type of heart disease during life despite repeated clinical, electrocardiographic, and roentgenologic studies, plus one cardiac catheterization study. He was considered a case of chronic myocarditis antemortem and called a beriberi heart by the pathologist at postmortem. In 1953, when first studied roentgenographically by upright-recumbent teleroentgenograms,

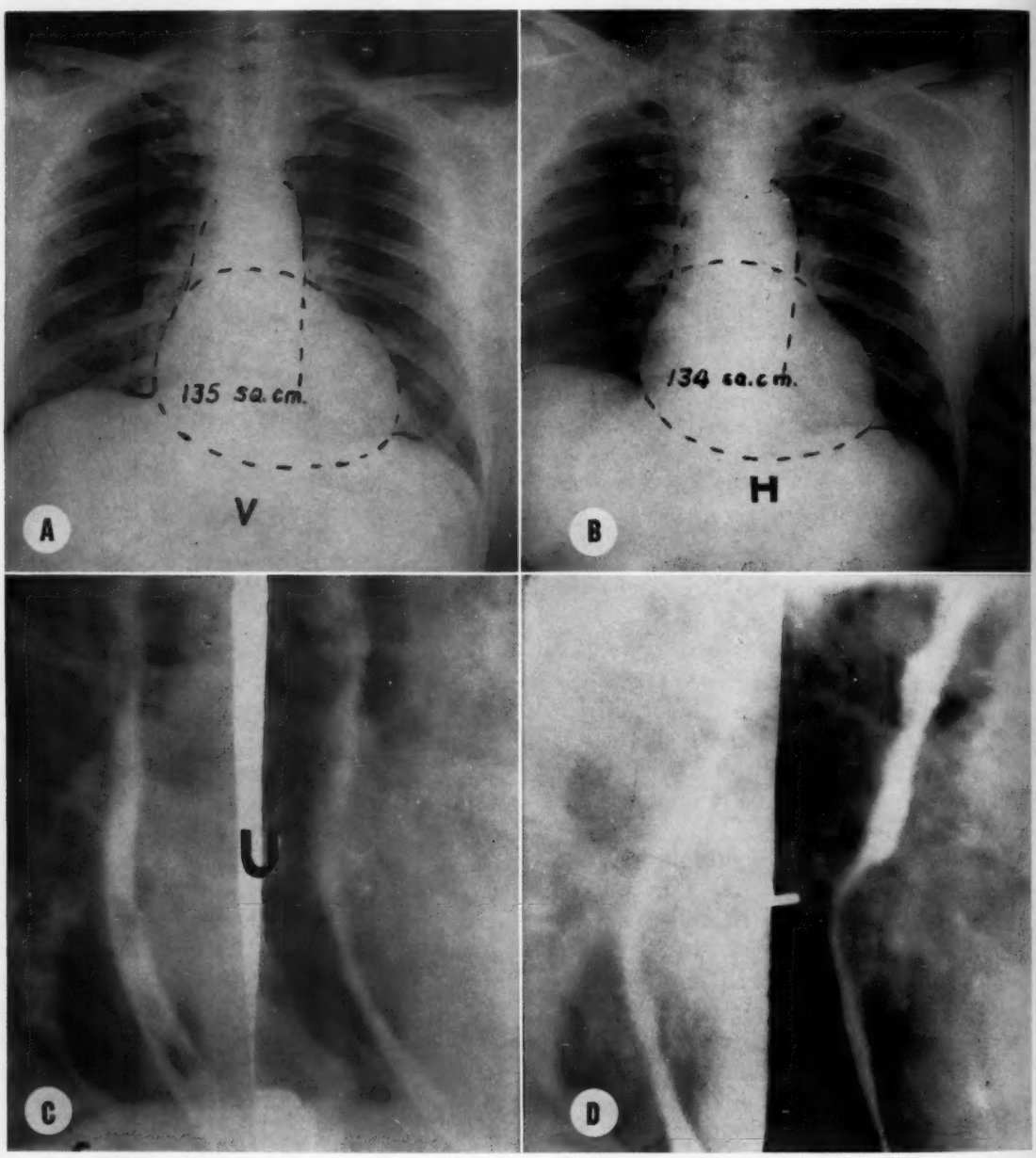

FIG. 6. W.H. Hypertensive heart disease, moderately severe. Male, aged fifty-seven. Height 68 inches, weight 163 pounds. Pulse standing 66, recumbent 66. (*A*) Upright teleroentgenogram. (*B*) Supine teleroentgenogram. (*C*) Upright right anterior oblique esophagrams. (*D*) Recumbent (supine oblique esophagram). No appreciable change in transverse diameter or frontal area measurement on upright-supine teleroentgenogram comparison. Upright esophagrams show normal configuration (*C*). Recumbent esophagrams (*D*) show localized compression and posterior displacement opposite left auricle level, considered a "false positive" for left auricular dilatation (no regurgitation murmur).

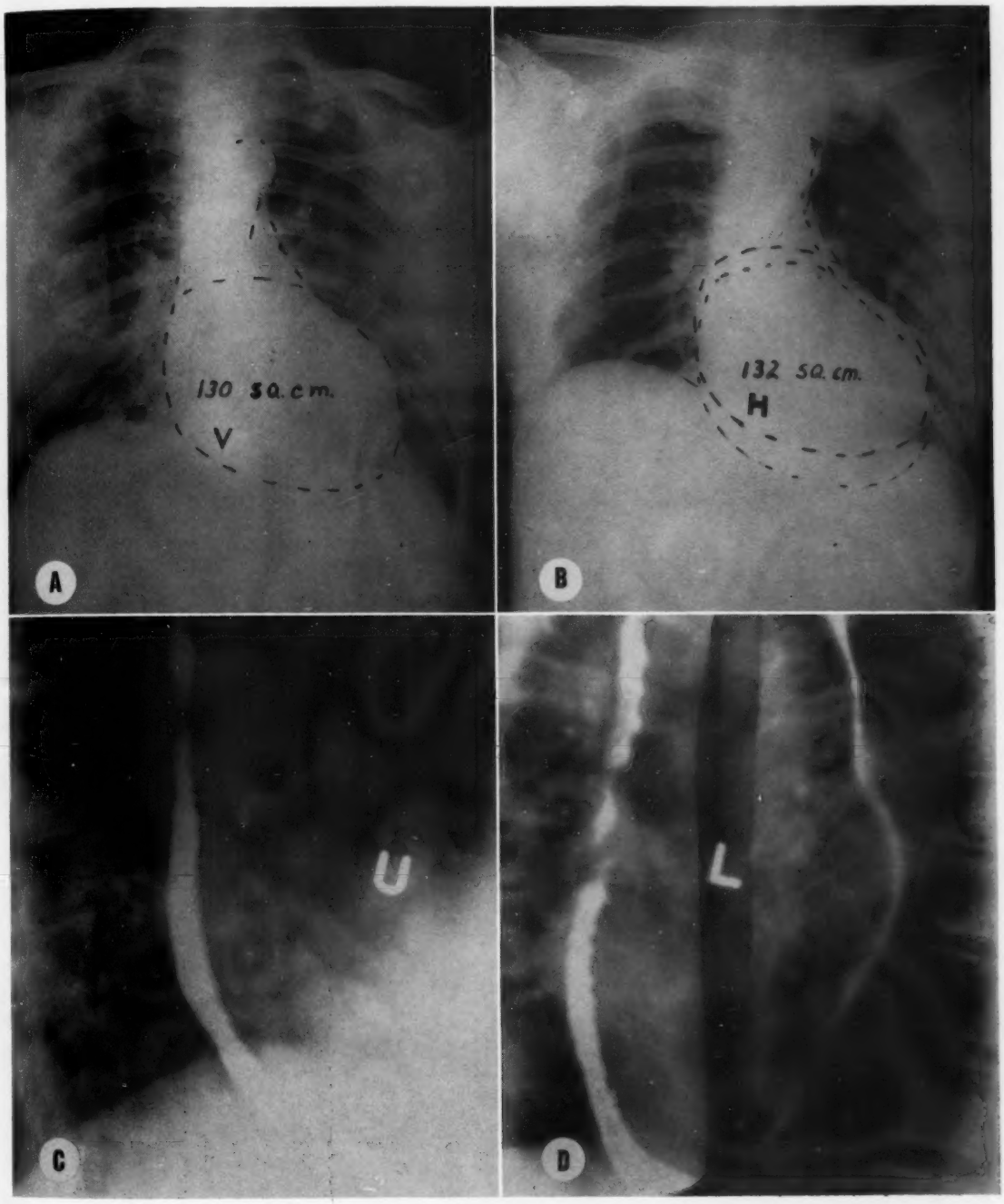

Fig. 7. C.M. Old myocardial infarction. Male, aged sixty-six. Height 68 inches, weight 137 pounds. Pulse standing 76, recumbent 64. (*A*) Upright teleroentgenogram. No significant change of transverse diameter or area on upright-recumbent teleroentgenogram comparison. (*B*) Recumbent teleroentgenogram (upright cardiovascular silhouette superimposed). (*C*) Upright esophagram normal. (*D*) Supine oblique (left) and prone oblique (right) esophagrams showing "false positive" posterior displacement opposite left auricle in prone oblique only.

he showed an increase in transverse diameter from 16.0 to 18.1 cm. and an increase in area from 156 sq. cm. to 182 sq. cm. (Fig. 9). Later, when his cardiac functional capacity was further impaired, change in size on change from upright to recumbent position was negligible, the transverse diameter change being from 19.8 cm. to 20.3 cm. and the area from 214 sq. cm. to 217 sq. cm.

In the 8 cases clinically classified as organic mitral valvular disease, uncomplicated, only 2 showed transverse diameter increases of more than 5 per cent and 2 showed decreases of 4 per cent (Table II, Group 1). Only 3 showed area increases of

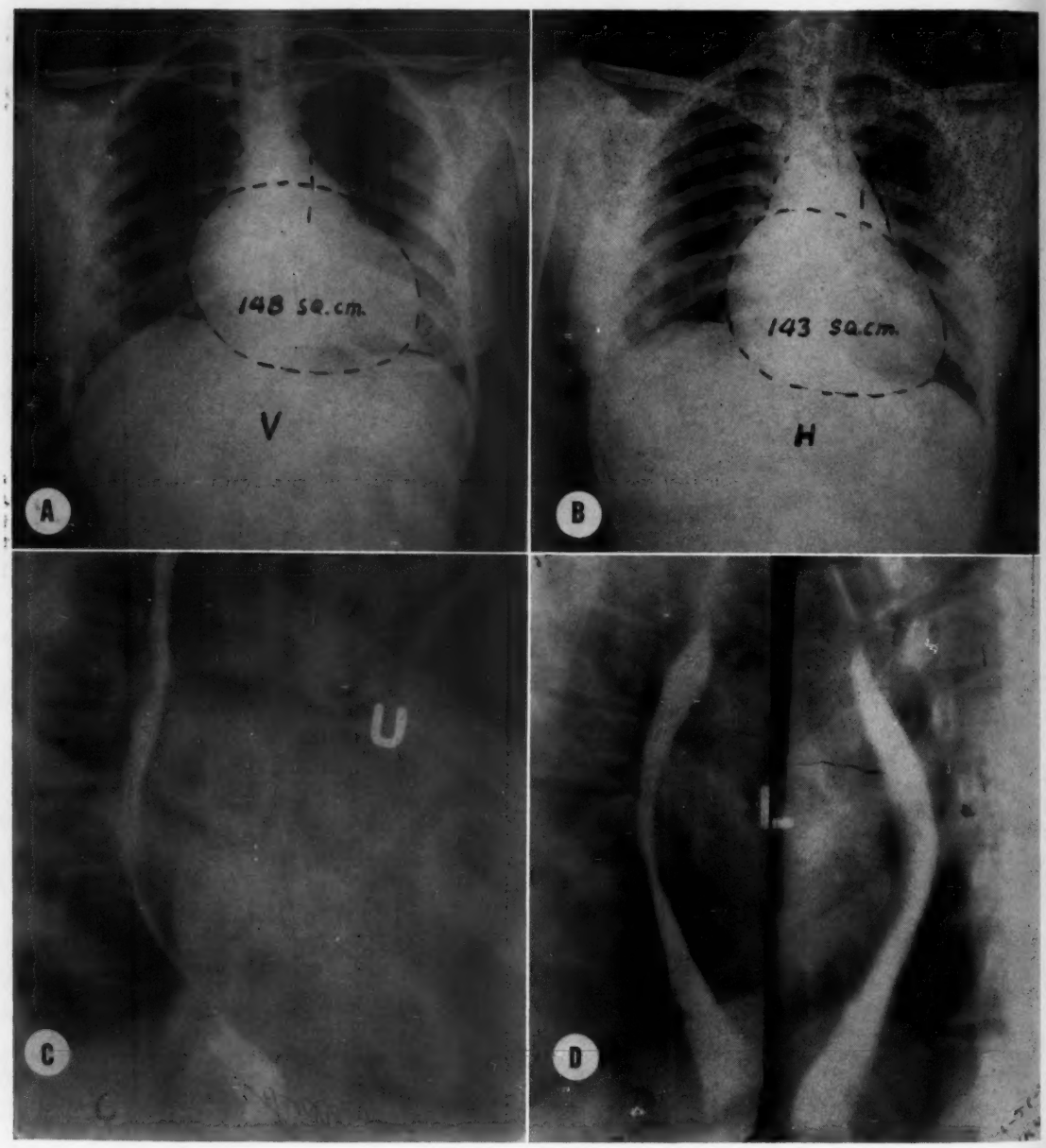

FIG. 8. H.S. Mitral stenosis, ten months post-valvulotomy. Female, aged thirty. Height 62 inches, weight 118 pounds. Pulse standing 88, recumbent 80. (*A*) Upright teleroentgenogram. (*B*) Recumbent teleroentgenogram. Very slight decrease in both transverse diameter and area on assuming recumbency. (*C*) Upright oblique esophagram. (*D*) Recumbent supine oblique and prone oblique esophagrams with no frank change in degree of compression and posterior displacement of esophagus opposite level of enlarged left auricle.

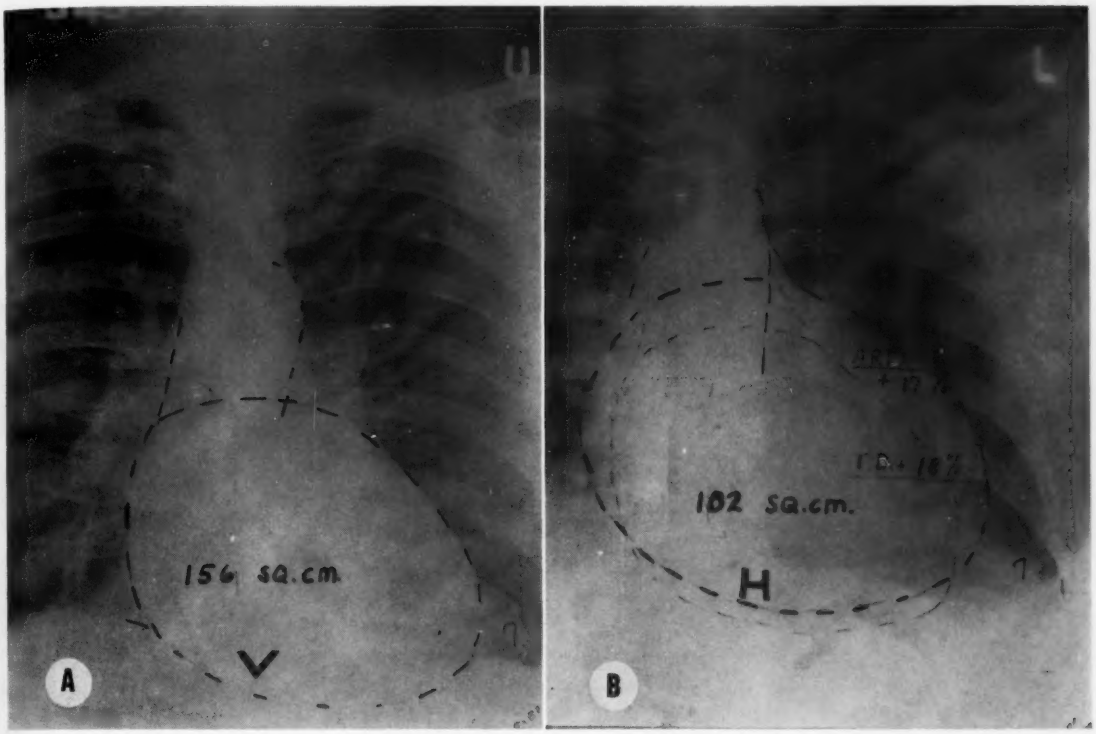

FIG. 9. S.D. Myocardial disease of undetermined etiology. Male, aged forty-six. Height 67 inches, weight 178 pounds. Pulse standing 100 (fibrillation), recumbent 100. (*A*) Upright teleroentgenogram. (*B*) Recumbent teleroentgenogram (upright cardiovascular silhouette superimposed) showing marked increase in transverse diameter (18 per cent) and area (17 per cent) on assuming recumbency. Postmortem findings favored vitamin deficiency state—beriberi heart—accepted with reservations by clinician. Only slight increase in degree of posterior displacement of esophagus with patient recumbent despite marked increase in heart size.

more than 5 per cent, and 1 showed a decrease of more than 5 per cent. It was expected that significant area increases might regularly be noted in the group, since it is the contention of Kjellberg that all chambers will be more filled with blood when the patient is recumbent and, therefore, minor degrees of left auricular enlargement be more regularly seen by recumbent esophagram study.

No group, normal or pathologic, consistently showed large changes in heart size as measured in this study, when changing from upright to recumbent positions, although the linear build, asthenic or hyposthenic built individual with a standing pulse of 90–120 most frequently showed a significant change (Cases 9, 10, 11, 16, 19, and 25) (Fig. 11). Even here the change was not a constant one, since several individuals of linear build with relatively "small" hearts and rapid standing pulses did not show the expected increase in frontal area when measured in recumbency. The cardiac apex usually lay a little higher in the chest and a little farther laterally, and the great vessel shadows were almost invariably somewhat widened in recumbency (Fig. 4). An exception to the usual slight shift upward and outward of the apex was seen in each of several instances of eventration of the left diaphragm with Magenblase. Under these conditions when assuming supine recumbency the apex moved somewhat downward and medially as the large gas accumulation below the left diaphragm moved from the cardia to the gastric antrum.

While both the normal and abnormal groups are small and perhaps therefore statistically not very important, even from this rather small series it seems established

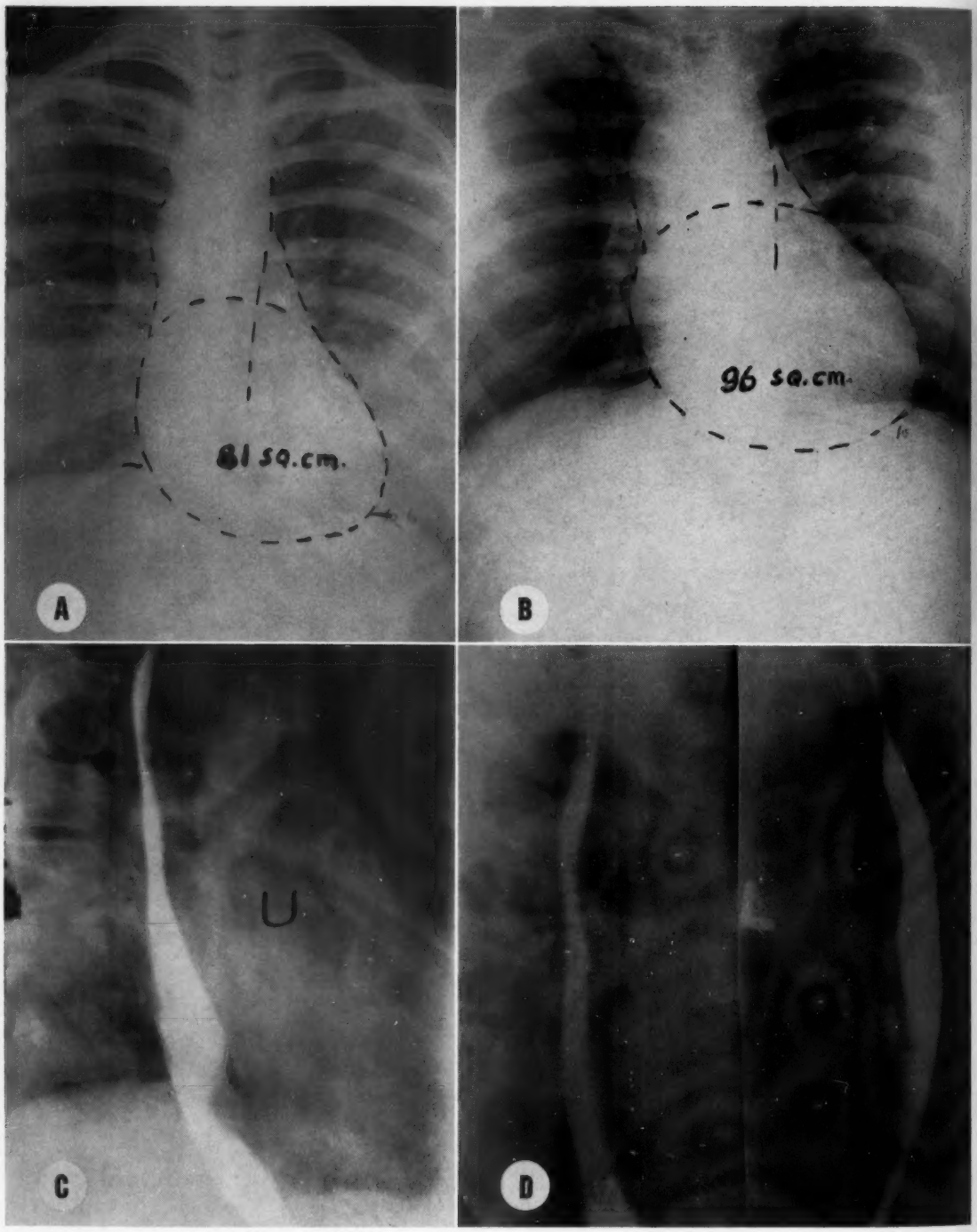

Fig. 10. D.P. Probable congenital heart (interventricular septal defect?). Female, aged twelve. Height 62 inches, weight 104 pounds. Pulse standing 72, recumbent 90. (*A*) Upright teleroentgenogram. (*B*) Recumbent teleroentgenogram. More than usual increase in transverse diameter and frontal area on assuming recumbency. (*C*) Upright oblique esophagram. (*D*) Recumbent (supine and prone oblique) esophagrams showing no appreciable change in course of esophagus on assuming recumbency despite moderate increase in heart size.

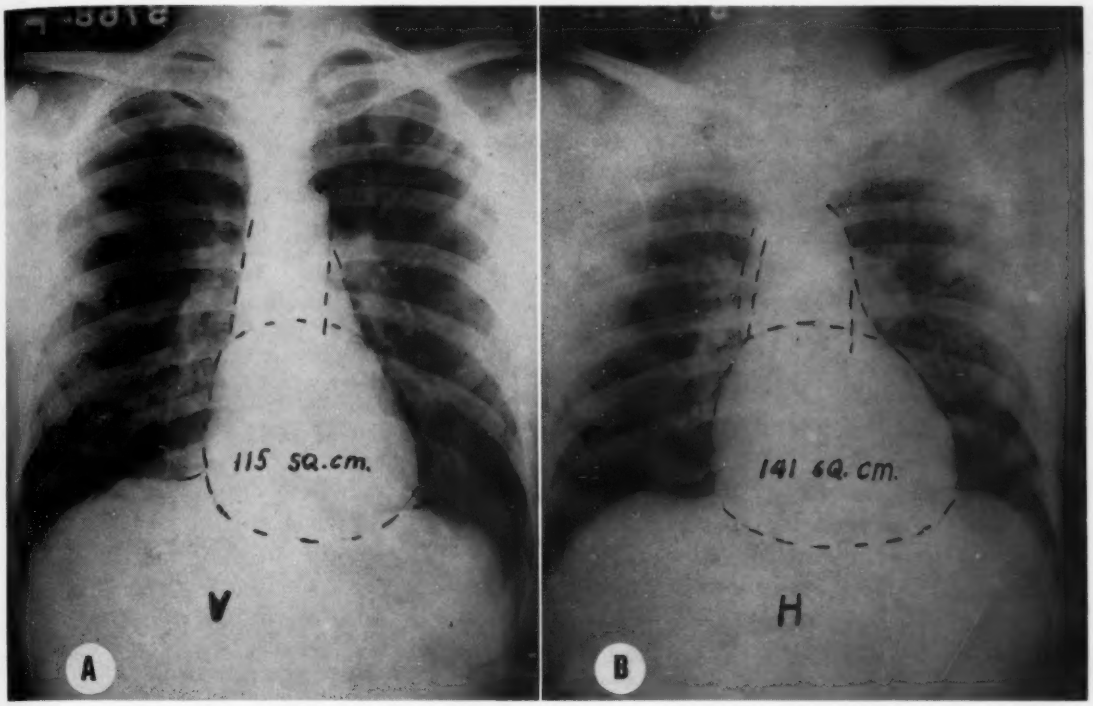

FIG. 11. W.S. Normal heart. Male, aged twenty-three. Height 72 inches, weight 166 pounds. Pulse standing 90, recumbent 72. (*A*) Upright teleroentgenogram. (*B*) Recumbent teleroentgenogram. Moderate increase in transverse diameter and frontal area on assuming recumbency. There was no change in the course of the barium-filled esophagus on comparing upright and recumbent position esophagrams.

that a significant enlargement is not commonly measured teleroentgenographically, the usual change both in linear and area measurements being a rather minor one. Nevertheless, an occasional significant and relatively large increase will occur in a normal as well as in a diseased heart. Hence, this possibility should always be considered when one must evaluate heart size with the patient in supine or prone position and apply standards which in this country have been established for upright techniques of orthodiascopy or teleroentgenography.

SUMMARY AND CONCLUSION

In a series of 70 patients of whom 25 were clinically and roentgenographically normal and 45 had clinically diseased hearts, standing anteroposterior and supine teleroentgenograms were made and the transverse diameter and frontal area measurements compared to determine the frequency and the degree of so-called "physiologic dilatation" occurring in recumbency.

The average increase in the transverse diameter was 4 per cent in the normal group and 2 per cent in the pathologic. The largest increase was 17 per cent in the normal and 13 per cent in the abnormal group.

The average increase in frontal area was approximately 4 per cent for the normals and approximately 3 per cent for the abnormals. The greatest increase in the normal group was 24 per cent, and the greatest in the abnormal group was 18 per cent.

A few instances of small decreases in both linear and frontal area measurements were encountered in both normal and abnormal hearts when the patient assumed the position of recumbency.

None of the above findings negate the concept of physiologic dilatation of the

heart occurring in some degree regularly in recumbency, since frontal measurement changes do not necessarily reflect volumetric changes. Nevertheless, the results would indicate that when roentgen methods are used, measurements in recumbency will frequently vary little from those in upright study, and this is particularly true of area measurements.

We were unable in this series to demonstrate a close correlation between changes in heart size on frontal projection teleroentgenograms made with the patient upright and recumbent, and changes in the course of the barium-filled esophagus. Cases showing the more marked degree of "physiologic dilatation" on assuming recumbency often showed little or no alteration in the course of the esophagus, while some cases showing no appreciable change in heart size showed pronounced changes in the course of the esophagus opposite the base of the heart, most of which were considered examples of "false positive" evidence of enlargement of the left auricle.

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VOL. 76, NO. 4

THE EXPANDING SCOPE OF CARDIOVASCULAR RADIOLOGY*

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TO DIAGNOSTIC radiology the last few years have brought numerous advances, not the least of which have occurred in examination of the cardiovascular system. The usual methods are confined to simple observations of the external contours, surface movements and calcification of the heart and vessels, and, as such, have limited diagnostic potential. More specialized methods allow detailed visualization of the interior of the heart, the entire arterial tree, the bulk of the peripheral and portal venous systems, some peripheral lymphatics and through their vascular channels, the parenchyma of several organs. The contrast techniques which provide such visualization and other highly informative procedures requiring radiologic assistance, such as cardiac catheterization and electrokymography, are commonly employed today in most medical centers.

No less expansive than this progress in methods are the realms of anatomy and physiology, both normal and pathologic, that have become accessible to the radiologist. Certain dynamic aspects of normal cardiac anatomy can be visualized for the first time. The pathologic anatomy and accompanying physiologic alterations of several cardiovascular lesions are as well, if not better, studied by these methods as by any other form of gross examination. Of no little importance is their clarification of the limitations and potentialities of the simpler roentgen studies.

Justifiable use of these procedures is limited to a minority of patients but a disproportionately large number of physicians becomes involved in their performance or in the wide range of new therapeutic procedures which they facilitate. The increasing requirement for skilled man hours and elaborate apparatus for diagnosis and treatment of a few patients has important economic and organizational implications.

Increasing, also, is the responsibility of the radiologist. The undertaking of certain major surgical procedures may depend upon the information he provides with these newer methods. Although generally safe they are not totally harmless[6,10,26] and present the undesirable situation of diagnostic roentgen examinations carrying the risk of morbidity or mortality. In this circumstance careful selection of patients and faultless performance with the best available equipment are demanded. Exposure of the patient to hazard for a non-informative study cannot be tolerated.

Unless we lose perspective it is pertinent to recall that few of these examinations are new in conception or performance. Dr. Scott's Carman Lecture of 1950 provides a splendid historical account of the origin of opacification techniques.[31] These began with peripheral venography and arteriography in 1923[2] and 1924.[5] Egas Moniz' development of cerebral arteriography in 1927 is well known,[13] but his contribution to the first successful right heart angiocardiogram in 1931 is less familiar.[14] A technique for visualizing both sides of the circulation was first described by Robb and Steinberg in 1938. Among other things 1929 is remarkable as the year of origin of translumbar aortography[9] and cardiac catheterization.[16] Succeeding years have added refinement of technique, better implementation and more frequent application to all of these. The only realy new procedures are portal[27] and splenic venography[1] and lymphangiography[22] which have developed since 1950.

Of the several factors contributing to the

* Presented at the Fifty-sixth Annual Meeting, American Roentgen Ray Society, Chicago, Illinois, September 20–23, 1955.
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emergence of these contrast studies, undoubtedly the most important has been the brilliant progress of cardiovascular surgery. The preoperative necessity of accurate and detailed anatomic and physiologic information continues to be the principal stimulus to the development and exploitation of these methods. No little credit is also due those who have provided safe injectable contrast materials, sufficiently sensitive film and adequate roentgenographic equipment. Lastly and not least we must acknowledge our slowly developing appreciation of the considerable tolerance of the human cardiovascular system to puncture, catheterization and perfusion. Although limited, it is nevertheless much greater than any except the visionary pioneers would have considered possible.

The scope of this paper permits little more than mention of the more interesting or practical applications of the contrast methods. Angiocardiography may be used to examine the intrathoracic veins and the vasculature of the lungs, to determine the vascular component of mediastinal masses and, of course, to study the heart itself.[11] Although the pulmonary arteries and veins are greatly modified by a number of common diseases, these seldom are investigated because of the adequacy of other methods and the non-specificity of the angiographic findings.[36] The estimation of the resectability of bronchial neoplasm on the basis of vascular involvement may be an exception but apparently is a seldom used indication.[34] A few conditions, nearly all congenital and quite uncommon, have unique arrangements of pulmonary vessels that may be recognized. These are agenesis of a lung, intralobar pulmonary sequestration, absent pulmonary artery, anomalous pulmonary veins, arteriovenous fistula and supravalvular arterial stenosis (Fig. 1). For these noncardiac applications simple manual peripheral venous injection of contrast medium and relatively slow serial filming are the simple technical requirements.

To date angiocardiography has offered little aid in the management of acquired heart disease with the exception of pericardial abnormalities and atrial tumors.[25] The differentiation of pericardial effusion from cardiac dilatation occasionally offers clinical and roentgenologic difficulty. This may be simply resolved by the observation of an abnormally wide radiolucent band between the heart cavities and outer border of the heart shadow. Two[41] unpleasant experiences with ventricular fibrillation during diagnostic pericardial tap, one fatal and the other requiring thoracotomy for resuscitation, have convinced our cardiovascular team that angiocardiography is the safer diagnostic maneuver. In the same way the method has also helped recently in the recognition of several clinically atypical cases of constrictive pericarditis. An especially important problem, the accurate preoperative assessment of the functional state of the mitral valve, has been approached and apparently with success in some quarters,[42] but we and others[37] have been disappointed in the ability of simple venous angiocardiography to distinguish between mitral stenosis and mitral insufficiency.

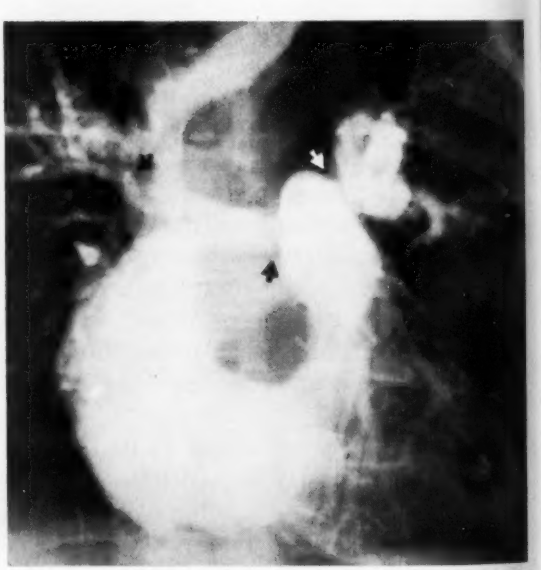

Fig. 1. Supravalvular pulmonary arterial stenosis (arrows). At three sites in the pulmonary arteries sharp pressure gradients were demonstrated by cardiac catheterization. Note ectasia of upper lobe arteries.

There is no question that congenital heart disease is the most frequent indication for angiocardiography. By peripheral venous injection and filming as slowly as two films per second, the physiologic defect can be established in nearly every anomalous heart, and, by meticulous attention to roentgenographic technique and projection, intimate anatomic details become visible. For instance, left to right shunts of all types are regularly recognized by recirculation of contrast medium through the lungs. In suitable projections diagnostic mixing defects are frequently noted in the chambers receiving the shunts, thus allowing more precise localization (Fig. 2). Shunts in the opposite direction, from right to left, are easily discovered by early opacification of the aorta. Again, in the proper projection the underlying defect can generally be detected because the contrast material does not become significantly diluted before reaching it. The anatomic detail of coarctation and aortic arch anomalies, at times so important to the surgeon, is not difficult to demonstrate. With the best technique this simple form of angiocardiography can thereby generally distinguish the several forms of pulmonic stenosis and guide their treatment (Fig. 3 and 4).

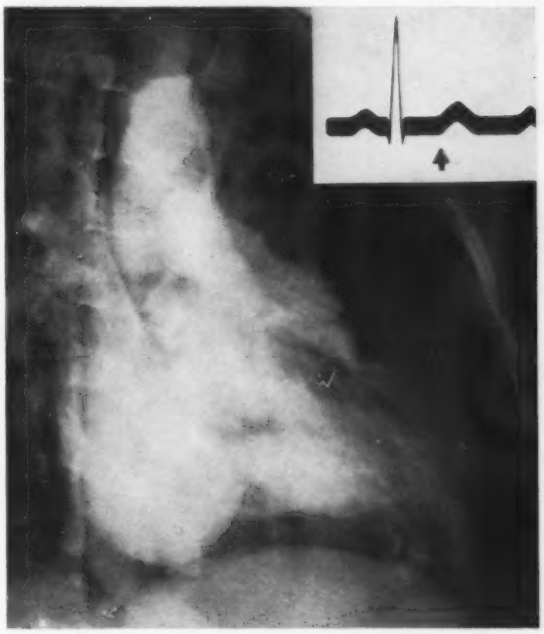

Fig. 3. Tetralogy of Fallot. Right anterior oblique projection. Infundibular stenosis (arrow) below normal pulmonary valves. Exposure during ventricular systole.

For more precise anatomic delineation

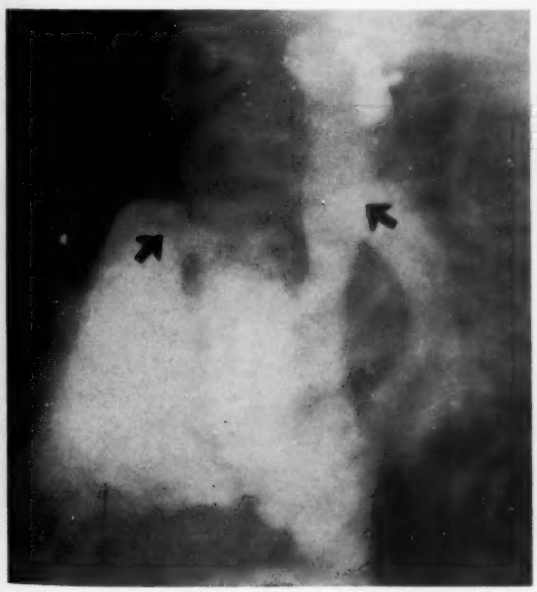

Fig. 2. Patent ductus arteriosus. Large dilution defect in the pulmonary artery between arrows.

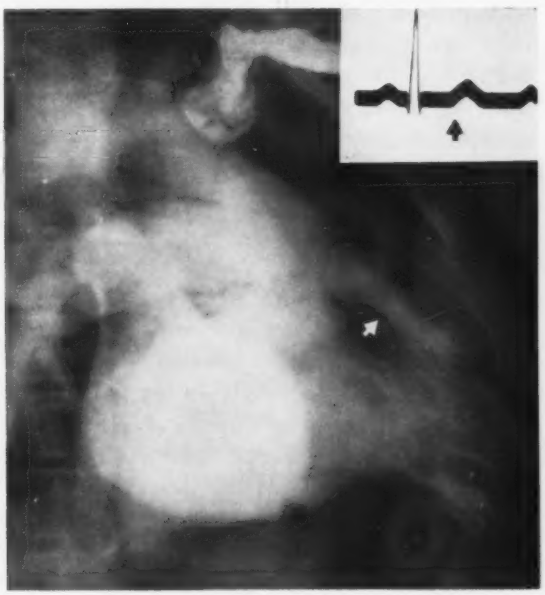

Fig. 4. Tetralogy of Fallot. Right anterior oblique projection. Pulmonary valve stenosis (arrow). Exposure during ventricular systole.

Fig. 5. Pulmonary valve stenosis. Selective injection in the right ventricle. (Printed through the courtesy of Sven R. Kjellberg, Karolinska Sjukhuset, Stockholm, Sweden.)

opacification of selected parts of the heart by injection through a cardiac catheter may be employed. The fullest exploitation of this method requires successful catheterization of the desired region, high pressure injection apparatus and simultaneous biplane filming at 6 to 12 films per second under general anesthesia with electrocardiographic control. The accomplishments of certain Swedish investigators[20,23] with this elaborate technique are remarkable (Fig. 5).

Opacification of the thoracic aorta or any one of its branches is quite feasible and may be obtained by injection through a properly placed catheter or by retrograde injection through needle or cannula in a large branch.[21] The anatomic details of coarctation and patent ductus are best seen by this technique and it may be useful in studying aneurysms before their surgical treatment.

Abdominal aortography is most widely used for delineation of occlusive disease of the aorta and its major branches[23] and for study of the parenchymatous organs, particularly the kidney, although the liver and spleen can also be evaluated through their arterial supply. For arteriography of occlusive disease translumbar aortic puncture under spinal or local anesthesia is the safest and simplest approach (Fig. 6). Only standard roentgenographic equipment is needed for adequate visualization of the entire distal arterial tree.[7,18]

In the same manner the visceral arteries become visible but better detail can be obtained by selective catheterization of their arterial supply and serial filming.[3] The principal indications for renal arteriography (and nephrography) have been the differentiation of renal cysts, tumors and other retroperitoneal masses, the detection of arterial anomalies and the assessment of renal blood supply relative to the choice of conservative or extirpative surgery (Fig. 7).[25,33] There is some discordance of opinion as to its real usefulness in directing surgical treatment.[28] Pelvic arteriography can assist the gynecologist in the detection of uterine and ovarian tumors[4] and the obstetrician in the search for ectopic pregnancy and the site of the placenta.[38] An interesting use recently described for arteriography is the localization of parathyroid adenomata.[32] The coronary arteries have been well shown by thoracic aortography[8] for other purposes but the procedure apparently has not been used extensively in older subjects who may have occlusive disease.

Peripheral and cerebral arteriography are often useful for the diagnosis and surgical treatment of arteriovenous fistulas, aneurysms, obstructions and neoplasms of these regions (Fig. 8 and 9). Percutaneous puncture and ordinarily available roentgenographic equipment are the simple prerequisites although serial filming is advantageous.[39]

The two major venous systems, systemic and portal, can be studied to advantage on special occasions but unlike the arterial tree, the direction of blood flow does not

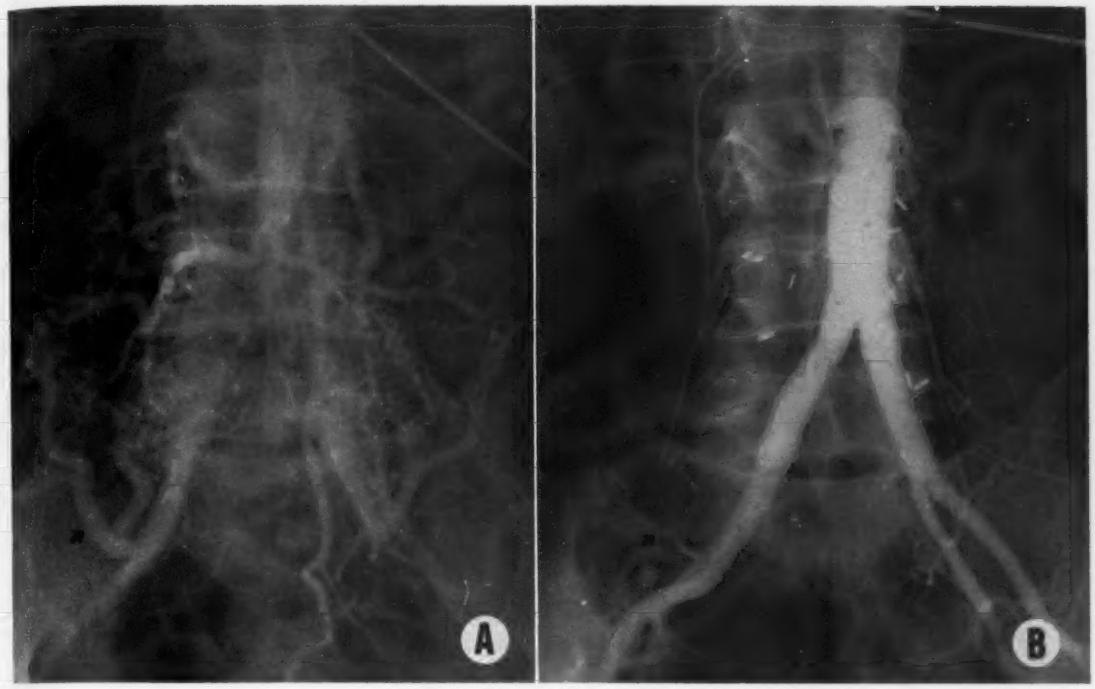

FIG. 6. Abdominal aortic occlusion. (*A*) Note collateral circulation through lumbar and iliolumbar arteries (arrows). (*B*) Three months after aortic and bilateral iliac end-arterectomy. Collateral vessels have returned to normal size.

FIG. 7. Normal renal arteriogram. Fortuitous selective injection of renal artery during translumbar aortography.

provide for visualization of all patent tributaries. The technical difficulties of reliable peripheral venography are reflected in the numerous techniques that are described. That the question of venous patency can be resolved by upright venography is convincingly stated by Greitz.[19] Roentgenologic assessment of venous insufficiency is more difficult. Smaller portions of the systemic venous system, pelvic, osseous and hepatic veins[40] and the dural sinuses[30] are accessible by special techniques but are seldom studied.

Portal venography is the latest of these contrast examinations to find a secure place in our roentgenologic armamentarium. It is accomplished by direct injection of a tributary at laparotomy or more conveniently by percutaneous splenic puncture.[15] Serial filming is a distinct advantage. Information of great value is provided concerning the recognition and treatment of the several types of portal hypertension

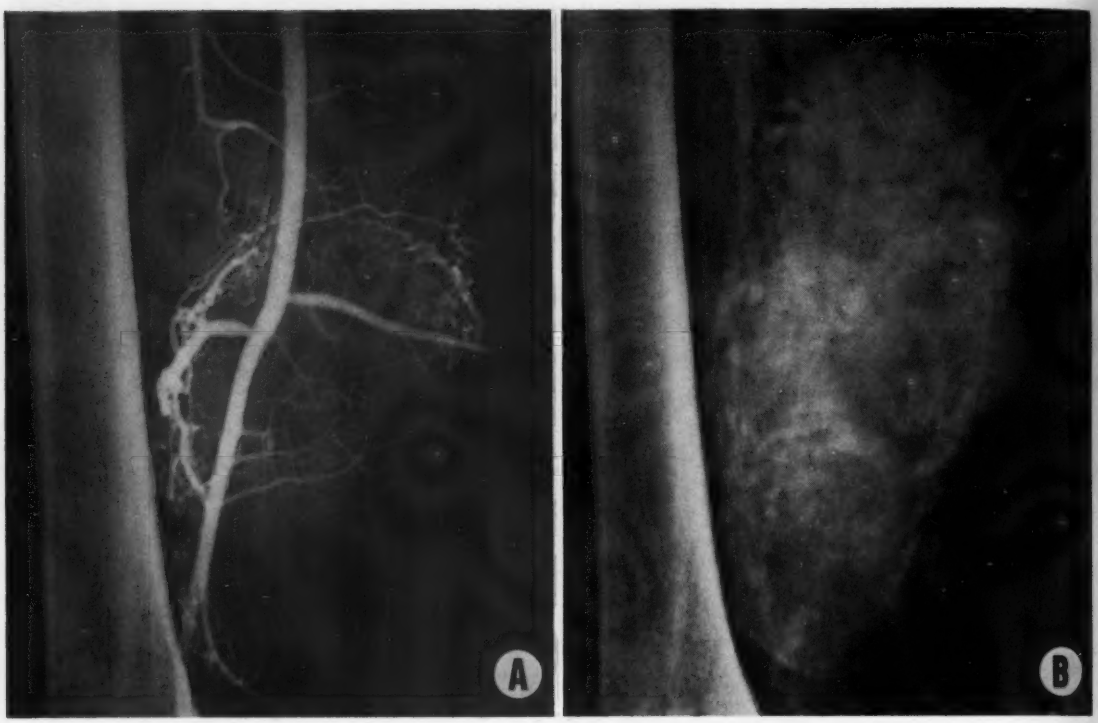

Fig. 8. Spindle cell sarcoma of thigh. Femoral arteriogram showing arterial supply (*A*), capillary and venous drainage (*B*). Preliminary clinical impression was femoral aneurysm.

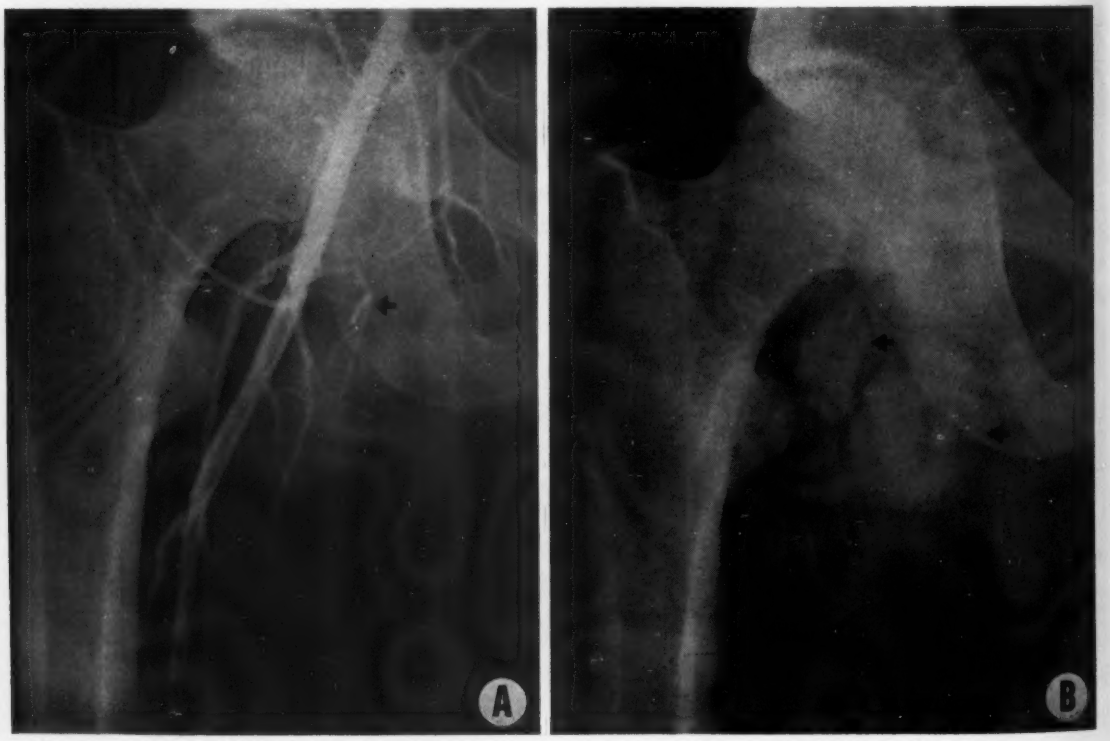

Fig. 9. Arterial obstruction. Complete superficial femoral and partial profunda femoris occlusion resulting in gangrene of foot. A small artery (arrow, A) provides opacification of enlarged lymph nodes (arrow, B).

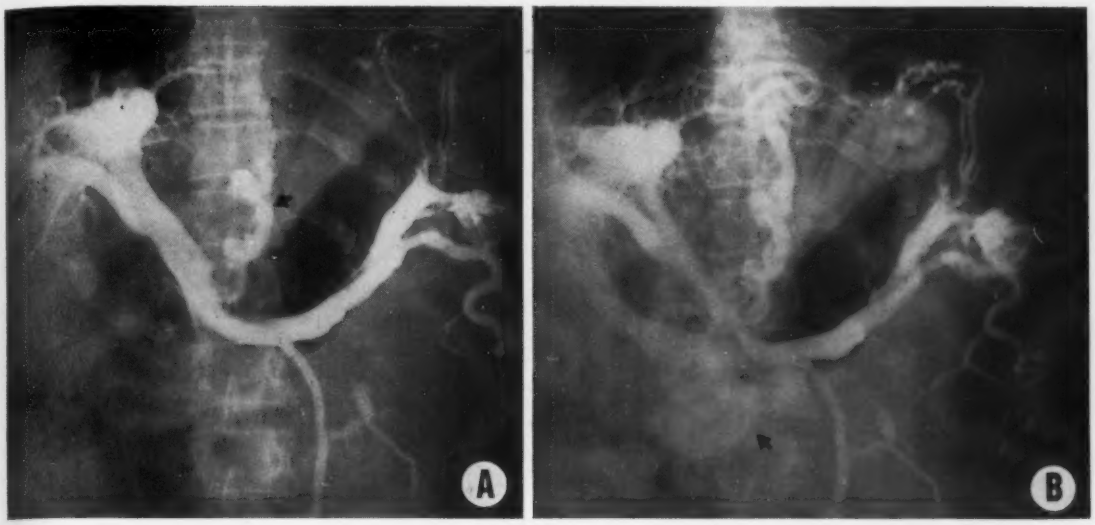

Fig. 10. Cirrhosis of liver. Splenic venography demonstrates small, deformed vascular pattern in liver, coronary vein leading to esophageal varices (arrow, A) and caput medusae (arrow, B) fed by periumbilical vein.

(Fig. 10). Considerable deformity of the veins is caused by mass lesions of the liver and pancreas, but it remains to be seen how this will assist the early recognition of these lesions.

This presentation, hardly more than a tabulation of the practical present day contrast examinations of the cardiovascular system can provide only a glimpse of this expanding field. What may we expect of the future?

In investigation of cardiac defects selective angiocardiography ought to find wider application. It is reasonable to expect that detailed anatomic and physiologic information, unavailable by simple venous angiocardiography, will be required as new intracardiac procedures are developed. Such a trend requiring greater skill, time and equipment may localize the procedure to larger medical centers. Mechanical problems have seemingly been largely overcome although Dotter[12] has recently indicated the desirability of shorter exposures. Intensification of the fluoroscopic screen will make cineroentgenography more widely available but this will be of little advantage unless sharp detail is maintained.

Similarly a trend toward selective visceral arteriography by properly placed catheter injection seems to be established. As yet the coronary, hepatic, pancreatic, intestinal and thyroid circulations, to name a few, are not adequately selectively studied. Perhaps the technique of injection distal to temporary occlusion, as described by Nordenström[29] for pulmonary arteriography, may have wider application. A somewhat similar "trapping" technique has recently been described for venography.[17] The control of the circulation that occlusion affords eliminates the need for high frequency filming.

Lastly, we may expect continued efforts to produce contrast materials that are harmless in massive amounts, completely non-irritating to the vascular endothelium and similar in physical properties to blood. Such ideal materials, in removing the disadvantages of present day media, would allow wider explorations in gross pathology and perhaps add a new tool to experimental physiology.

Broadly considered, cardiovascular radiology today includes many organ systems other than the heart, and, in contributing to the diagnosis and treatment of diseases of these, it becomes of interest and service to many branches of medicine. Recent progress has been exciting and promises

to continue. In the pride of accomplishment at a relatively satisfactory level contemporary radiologists may forget those early investigators who pointed the way to a new radiologic dimension of human anatomy, the vascular lumen.

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ACCOMPLISHMENTS AND POSSIBILITIES IN CARDIOVASCULAR SURGERY*

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THE contributions of the physiologist and radiologist have been the foundation for the rapid and dramatic progress which cardiovascular surgery has made in recent years. Each of these specialties has accelerated the pace of the other. The physiologists and the cardiologists have clarified the pathologic physiology of many cardiac abnormalities, have helped to evaluate the results of surgical procedures, and helped find means to enable patients better to withstand the operative insult. Radiologists have been heavily relied upon for a diagnosis. In fact, there are few cardiovascular abnormalities which do not require radiologic aid in diagnosis. In turn, radiologists have expanded their diagnostic horizons by new techniques of contrast visualization. No doubt no one of these specialized endeavors would have made such advances without the needs, demands, and contributions of the others.

On reflection this progress has been tremendous. As medical students Dr. Figley and I marveled at the then small field of cardiovascular surgery, the gamut of which was almost covered by watching Dr. Gross ligate and later divide a patent ductus. We had memorized by rote the defects in the tetralogy of Fallot, and would never have guessed that someday someone would write a paper on a means of estimating the size of the orifice of the mitral valve from data of cardiac catheterization. I remember vividly on changing services as an interne on November 1, 1944, how Drs. Blalock and Taussig deliberated over the care on the first postoperative day of a certain patient. She was the first of a series numbering now over 1,350 patients operated upon by Dr. Blalock and his assistants for pulmonary stenosis. I believe this contribution more than any other single one opened the field of cardiovascular surgery, stimulated physiologists and radiologists to investigate these lesions, and enticed keen, bright minds, returning from the wars, into the new field of cardiovascular surgery.

Even though I would like to give a comprehensive review of cardiovascular surgery, I must get down to specific examples to show where we have been and where we hope to go in this field.

The first congenital cardiac defect treated surgically was so well done by Gross that few would argue against the complete validity of this method. Most of us will agree that the best treatment for patent ductus arteriosus is to interrupt it. An argument still smoulders between those who advise ligation and the advocates of division. It seems to me that this is largely a matter of personal preference.

In patients with patent ductus one of the problems which plagues us is the handling of those who have pulmonary hypertension. Pulmonary hypertension usually occurs under one of two circumstances. Certain infants and children with patent ductus are found to have a pulmonary arterial pressure approximating the systemic pressure. It would seem to represent a persistence of a fetal state in which the two pressures are essentially the same. Corrective operation is urgently indicated before permanent changes occur in the lung. In these patients with pulmonary hypertension the diagnosis is most difficult, for there is no continuous gradient of pressure between the systemic and pulmonary circuit, and the characteristic continuous machinery murmur is usually lacking. We rely heavily on radiologic aid and data from catheterization in the accurate diagnosis in these patients.

* Presented at the Annual Meeting of the American Roentgen Ray Society, Palmer House, Chicago, Illinois, September 20–23, 1955.
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The other group of patients with pulmonary hypertension and a patent ductus arteriosus are usually older, teen age or over. We do not know whether pulmonary hypertension has persisted from birth or if it is the result of continued increased pulmonary blood flow. In this group one may find a reversal of flow so that pulmonary arterial blood enters the aorta. Since the ductus enters the distal arch, the toes may be cyanotic while the fingers and lips are normal. There is considerable doubt about which of these patients should be treated by interruption of the ductus. If left alone they develop progressive right sided failure and die shortly. Will the patient survive interruption of the ductus? Are the changes in the pulmonary vascular bed reversible? These are difficult questions. Most experienced workers would agree that the ductus should not be interrupted unless one can demonstrate that the preponderant shunt is from aorta to pulmonary artery at least some of the time. Patients with patent ductus and apparent reversal of flow from the aorta to the pulmonary arteries have survived operative intervention, but the majority succumb shortly after operation. The problem of patent ductus with pulmonary hypertension continues to be vexing and difficult both in diagnosis and in treatment.

Another congenital malformation, namely, coarctation of the aorta, has offered challenges with significant accomplishments by physiologists, radiologists, and surgeons. The radiologists with the aid of new developments in techniques have demonstrated so beautifully the pathologic anatomy of coarctation of the aorta. Cardiovascular surgeons have relieved most patients of hypertension and the ever present threat of rupture of the aorta or intercostal aneurysms. They have also used coarctation as a spring-board to investigate the problem of aortic grafts which can be used for the treatment of other aortic lesions. To the physiologists the condition furnishes a good clinical experiment to study a form of hypertension which can be cured by a single specific act. A definitive explanation for the hypertension which occurs in the upper part of the body in this condition has not yet been given. There is, to my way of thinking, almost overwhelming evidence that in many instances the kidneys play a dominant role in this elevation of pressure.

Coarctation of the aorta is a disease which is readily diagnosed if one simply considers it and feels for femoral pulses or evidence of collateral circulation about the chest. The obvious and ideal treatment is excision and re-anastomosis of the aorta. When the repair accomplishes a normal or near normal aortic caliber, then the blood pressure can be expected to return to normal levels. This has been true in all but 2 exceptional cases of the 200 we have treated, and in both we suspect a strong emotional element and the appearance of essential hypertension.

There are a number of circumstances which prevent such a simple primary anastomosis (Fig. 1). These include aneurysm of the aorta or of the adjacent intercostal arteries, a long segment of coarctation so that the adjacent aortic ends cannot be approximated, a small proximal aortic segment or advanced disease of the adjacent aortic wall. For these conditions we use an arterial homograft as Gross has suggested. Since we have grafts routinely available and since the atypical low thoracic coarctation can be suspected on the basis of clinical findings, we do not routinely employ angiocardiography or aortography in patients with coarctation of the aorta but reserve this for those in whom the picture is not typical. In our experience only about 5 per cent of the cases have required a graft.

Arterial grafts and methods of procurement, sterilization, and preservation have been studied intensively. When it was established that the graft dies after implantation, it was a natural step to use a synthetic material, such as nylon, orlon, or dacron which can be easily procured, positively sterilized, and is inherently stronger than a homograft. Investigation is being conducted in many centers currently and it is

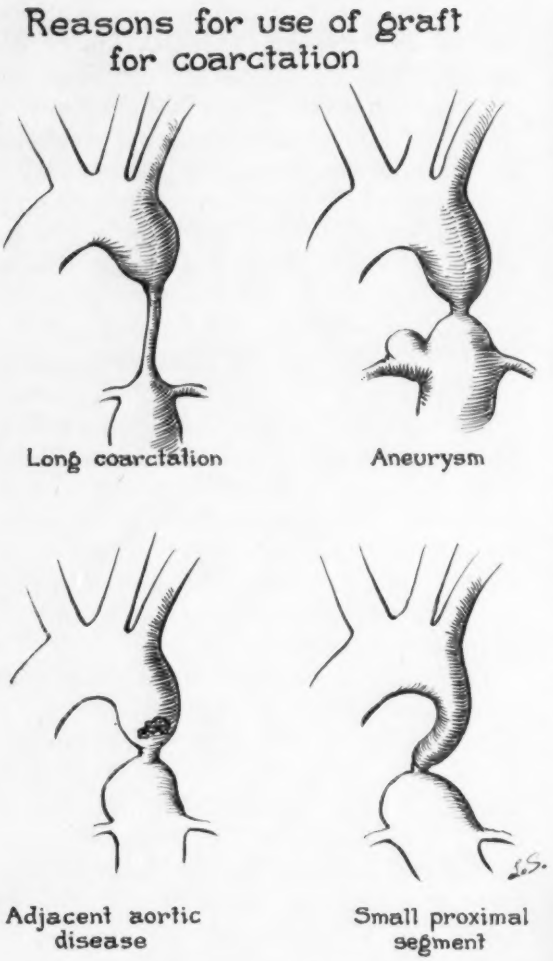

Fig. 1. Conditions encountered with coarctation of the aorta for which grafts are required.

probable that in the near future one may order a fitting for any portion of the vascular tree much as one orders pipe fittings at the present time.

Success in the treatment of coarctation of the aorta has encouraged the surgical treatment of other lesions of the aorta and great vessels. Aneurysms presented a challenge because of the great risk to the patient and the fact that in many instances the remaining aorta may be functionally sound. I have been surprised to see how often syphilitic aneurysms are saccular and represent localized blowouts with a relatively narrow mouth (Fig. 2). One may isolate and clamp the neck of such an aneurysm, excise it, and suture the aorta with standard technique (Fig. 3). In other instances where the entire wall of the aorta is diseased, the aorta may be occluded while a graft is sutured in place. One of the best and most common examples of the use of a graft for an aneurysm is for the arteriosclerotic aneurysm, which is almost invariably located in the abdominal aorta distal to the origin of the renal arteries (Fig. 4 and 5). Here occlusion of the aorta for a period up to several hours does not result in permanent damage. More proximally the aorta may be safely occluded only for a brief period which becomes briefer as one approaches the heart. In the thoracic aorta and distal arch, one may often occlude the aorta for a sufficient period of time to allow insertion of the graft. On the arch itself, however, this may not be done and some form of a shunt graft is required during the aortic occlusion. In the case illustrated a graft was sutured to the ascending and the upper thoracic aorta following which the aorta was occluded and the aneurysm excised (Fig. 6). In this particular instance it was not necessary to re-anastomose the vessels to the head and arms as we had initially planned (Fig. 7). This type of an-

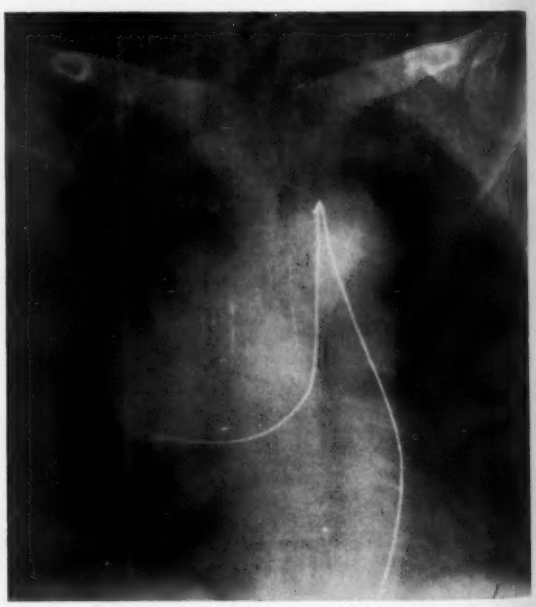

Fig. 2. Aneurysm of the ascending aorta opacified through a polyethylene catheter inserted percutaneously through the femoral artery. (Reproduced with permission from *Ann. Surg.*, 1953, *138*, 379.)

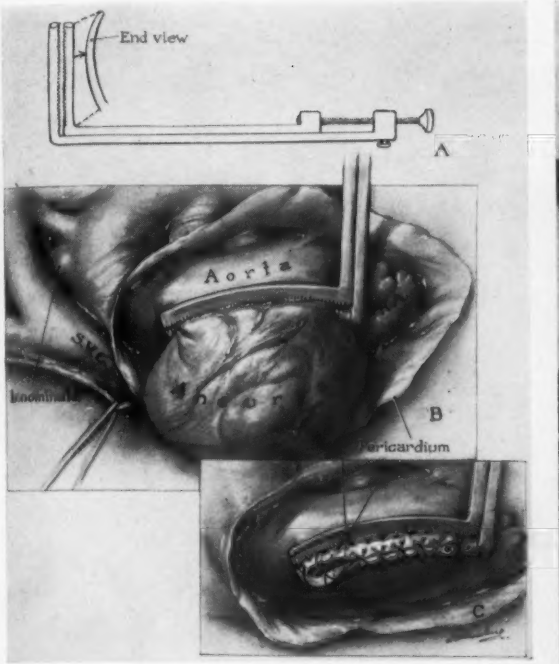

Fig. 3. Excision of aneurysm on the ascending aorta by isolation of the neck, clamping, and suturing.

Fig. 4. Excision of abdominal aortic aneurysm. The adjacent aorta and iliac vessels are mobilized and clamped. The aneurysm can more easily be dissected after division of the aorta. (Reproduced with permission from *Ann. Surg.*, 1953, *138*, 382.)

Fig. 5. Reconstruction of aortic continuity by homograft after excision of abdominal aneurysm. (Reproduced with permission from *Ann. Surg.*, 1953, *138*, 382.)

eurysm, namely one in the aortic arch, represents a major challenge in cardiovascular surgery.

Similar advances have been made in the treatment of occlusive disease of the aorta and great vessels, frequently manifested by the Leriche syndrome. Replacement of a thrombosed segment of the terminal aorta and iliac vessels, particularly in a young man, may result in remarkable improvement in the patient's walking distance, and complete rehabilitation (Fig. 8). Certain beneficial side effects are not to be taken lightly as impotence is a common complaint of these people. We have known wives who complained of their husbands' impotentia to reverse their complaints following operation.

Before proceeding with the newer and

Fig. 6. Aneurysm of the aortic arch. A homograft has been sutured to the side of the ascending aorta as a shunt graft.

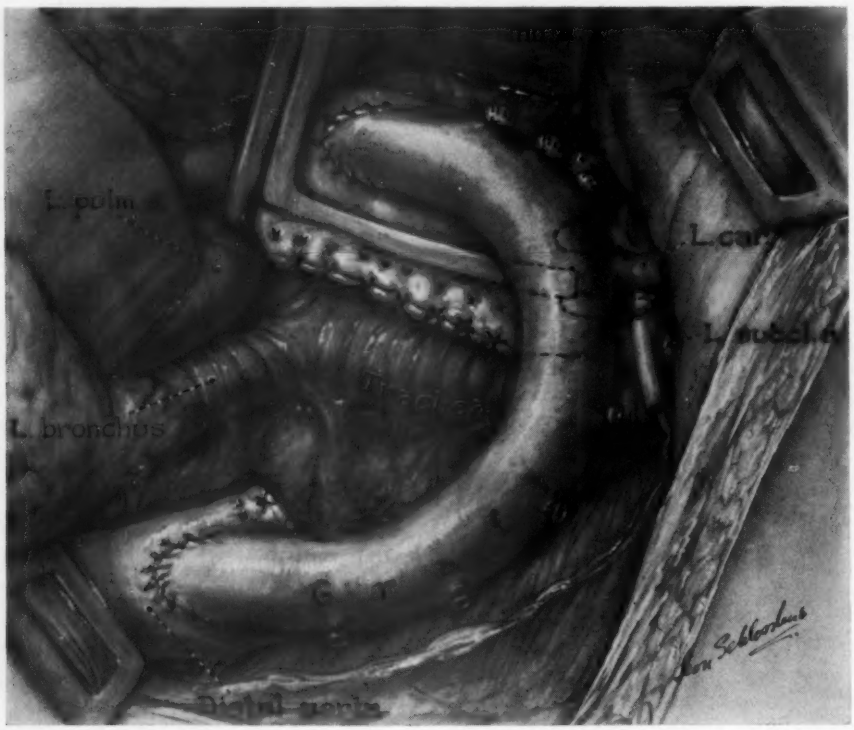

Fig. 7. Excision of aneurysm of the arch. With the shunt in place and open the aortic arch was clamped and the aneurysm excised. In this instance the vessels to the head and neck were not involved in the aneurysm and did not require anastomosis to the graft.

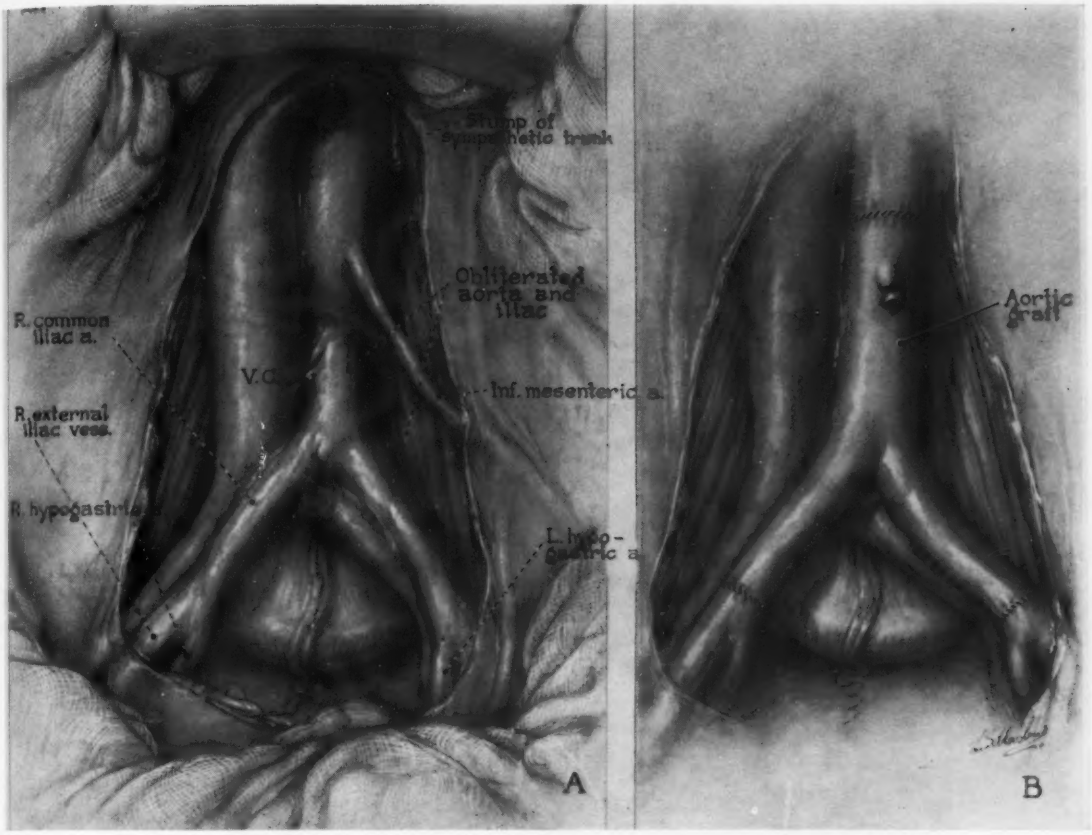

Fig. 8. Excision of aortic and iliac thrombosis and reconstruction by homograft.

perhaps more dramatic field of surgery inside the heart itself, a few words should be said about the treatment of constrictive pericarditis, as the surgical treatment of this disease was the first widely accepted operation upon the heart. The question as to which area of constriction is functionally more important appears to have been decided in favor of the ventricles. Almost everyone agrees now that constriction of the great veins and auricles is a rare cause of the clinical picture and that the ventricular encasement is dominant. Physiologic studies have elucidated this fact by showing evidence of elevated left atrial pressure as a result of inadequate left ventricular output. The other recent advance in the treatment of this condition is in the management of acute episodes of tuberculous pericarditis. Control of the infectious process by antibiotics has allowed early operation before the damaging effect of prolonged constriction has made itself felt upon the heart.

The most enchanting era of cardiovascular surgery lies ahead as methods are developed for operations within the chambers of the heart itself. In my opinion, in cardiovascular surgery, we are now in the same position as the late Walter Dandy when he was beginning his work in neurosurgery. His working credo was that nothing in neurosurgery was being done properly or could not be greatly improved. I doubt if many of the intracardiac techniques which we are now using at present will be similarly used in 1965. Numerous recent contributions suggest that we are approaching a turning point. Means have been developed for supplanting the function of the heart and lungs while the heart is open. Dr. John Gibbon has perfected his mechanical pump-oxygenator to the point where it is quite safe for use in animals. He has been

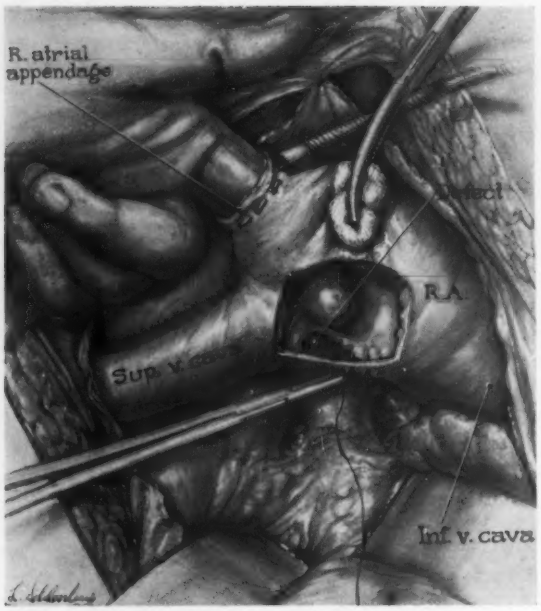

Fig. 9. Method of closure of atrial septal defect by suturing wall of the atrium to edge of the defect.

able to close an auricular septal defect in an adult with total by-pass of the heart and lungs. The group at the Mayo Clinic with Dr. Kirklin have modified and improved this apparatus and have operated upon 27 patients with 15 apparent cures of complicated defects. The group at the University of Minnesota with Lillehei, Varco, Cohen, and Warden have used several means for pumping and oxygenating, the most widely known of which is the cross circulation technique. By using a healthy adult for an oxygenator and side tracking a small and expendable portion of his cardiac output, a reduced flow of blood can be pumped to the patient and the function of his heart and lungs replaced. The rather complicated circuiting and slight hazard to the donor are far from ideal, but this technique has allowed survival and apparent cure of many types of defects. In addition, an im-

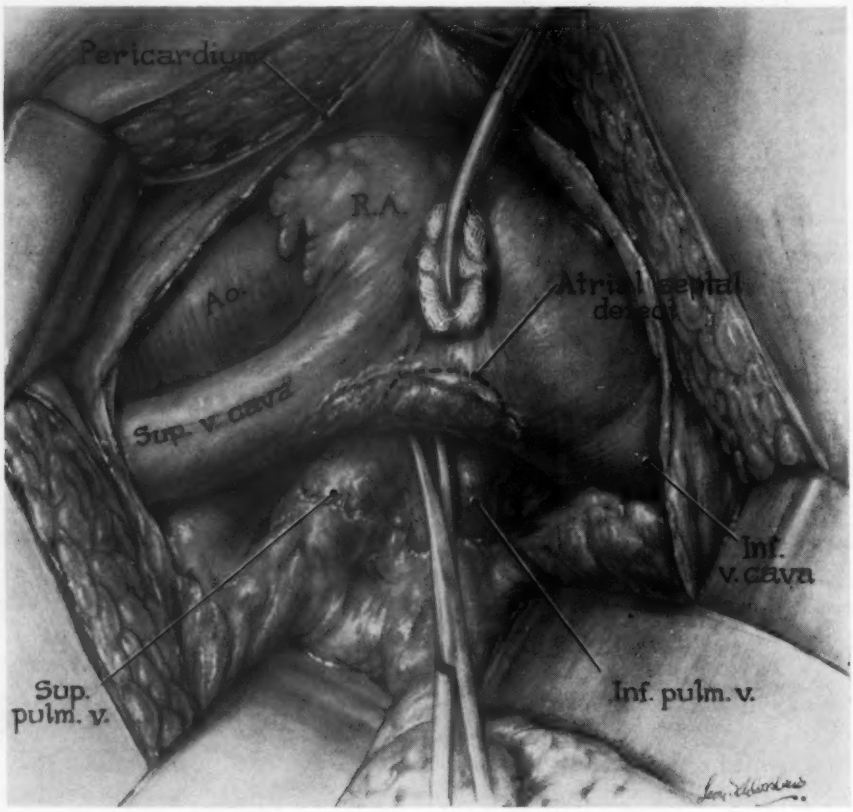

Fig. 10. Dissection of groove between right atrium and right pulmonary veins for atrial septal defect.

portant fact has been established; namely, that a relatively small volume of blood is required to maintain life during interruption of the circulation. Certainly a volume equal to the normal resting cardiac output is not required as most had previously thought. This approach has been a real stimulus to many investigators.

Another important development in intracardiac surgery is the use of hypothermia. The reduction in cardiac output, oxygen consumption and general metabolism with reduced temperature allows total interruption of the circulation for a short time. Much remains to be learned about hypothermia, however. A principal difficulty is the prevention of ventricular fibrillation which may occur with moderate cooling.

Hypothermia has been used by many to allow closure of interauricular and interventricular septal defects, treatment of pulmonary stenosis, and removal of a left auricular myxoma. There is little doubt that hypothermia will be used in some form in the future, but its exact status has not yet crystallized.

Intracardiac procedures which will command most attention are those for the repair of septal and valvular defects. Atrial septal defects have been closed by several methods. Swan and Lewis, using hypothermia, occlude the venae cavae, open the right atrium and suture the defect directly with excellent results. Most surgeons in this country, however, use one of several closed methods. Most involve intracardiac palpation with a finger inserted through the auricular appendage, and various techniques of suture of the defect or of the atrial wall into the edge of the defect (Fig. 9). In our experience the easiest technique is that of Bjork and Crafoord. They dissect the groove between the right atrium and the right pulmonary veins until the edge of the defect is encountered (Fig. 10). A purse-string suture may then be placed by starting at the base of the aorta and passing it through the septum. The other end is passed behind the superior vena cava and

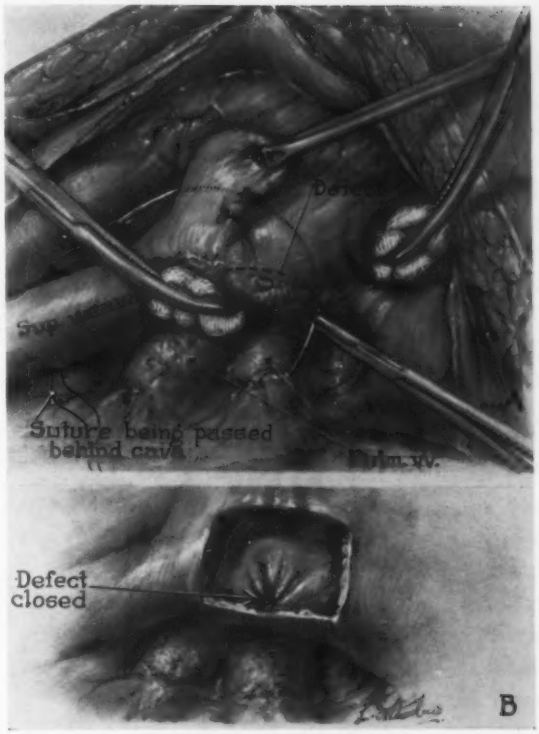

FIG. 11. (*A*) Placement of purse-string suture by needle passed through the septum. A finger is used in the auricular appendage for guidance. (*B*) Purse string passed behind superior vena cava and tied.

tied in the dissected groove (Fig. 11). This effectively closes the defect quite readily in almost all instances.

A special problem is encountered with an atrial defect when the defect is low near the atrioventricular valves, commonly called a defect of the septum primum. Here the suturing must be quite exact because of the adjacent atrioventricular valves and the conduction mechanism. The mortality has been high in all hands.

Though I personally favor, for the present, one of the closed methods, I am convinced that it is not the ideal treatment, and as soon as we can supplant the heart and lungs, an attack by direct vision will yield better results. We, thus far, have been able to close successfully 10 cases of typical atrial septum secundum defects. None of the 4 patients with septum primum defects, the low-lying ones, which we have explored

has survived. There is much work to be done.

The ventricular septum has not been as accessible as the atrial and has not yielded so easily to surgical attack. Reasons for this include the usual location beneath the aortic valve, where suturing must be exact to avoid aortic and tricuspid valves, the danger of air embolism to the aorta, the fact that hypothermia renders the ventricles more irritable and the risk of incising them greater, and the lack of a convenient way to feel within the ventricle as one is able to do in the atrium. Defects have been closed by various surgeons, some using hypothermia but most with some means of perfusion. The Minneapolis group uses cross circulation, that from the Mayo Clinic a total heart-lung machine, H. William Scott, Bailey, and others have used hypothermia, and Murray, Cooley, and others have used a blind suture. This problem is a principal target of investigators working towards open cardiac surgery as the defect is common, embarrassing to the patient, and itself a simple surgical problem—if one can make open cardiac surgery safe.

One of the most common of the intracardiac abnormalities which can be helped at the present time is the tetralogy of Fallot. The safest treatment at present is by an extracardiac procedure, the Blalock operation or a systemic-pulmonary shunt. Results with this operation have been gratifying as about 75 per cent of the patients are considerably improved. Many children are able to lead essentially normal lives, though a few have returned for a second operation on the other side to keep up with their growth. These good results must be borne in mind when one attempts an open intracardiac procedure for tetralogy of Fallot. Such curative procedures have been performed satisfactorily by the use of both hypothermia and perfusion techniques, and it is almost certain that in the future more such procedures will be performed. These few successes have shown that one can relieve the pulmonary stenosis by excision of the constricting bar of scar and muscle and close the defect. Overriding of the aorta presents problems which may require the use of prosthetics in some individuals.

Valvular pulmonary stenosis with an intact ventricular septum, often called pure pulmonary stenosis, can be quite satisfactorily treated in many instances by a blind procedure, which involves cutting or dilatation of the pulmonary valve, the knife being inserted through the right ventricle or the pulmonary artery. Swan has aggressively attacked this lesion by occluding the venae cavae, opening the pulmonary artery and accurately incising the pulmonary valve. His review of others and his own results show clearly that cutting of the valve under direct vision is necessary if one hopes to relieve completely the pressure gradient between the right ventricle and the pulmonary artery. Most of us have not completely relieved the pressure gradient although clinical results have been good in most instances. This, I believe, is one of the striking examples of a lesion which many surgeons are treating but which few are treating ideally. Two or three small incisions in the valve at just the right place under direct vision can usually restore the circulation entirely to normal.

The treatment of acquired valvular disease is so well known that I need make few comments. The continuing good results in the treatment of mitral stenosis nearly equal the hopes and expectations of surgeons and far exceed the hopes of most of our medical colleagues. Three-fourths of the patients have been greatly improved, about 15 per cent only moderately improved, and 10 per cent worsened or failed to survive. In many instances the commissures can be so split that an essentially normal valve mechanism results. None the less, if one had a safe means of supplanting the pumping action of the heart, in many of these patients one could much better open the mitral valve if direct vision could be obtained. By safe, I mean a procedure comparable to tracheal intubation and control of ventilation as is commonly done with

thoracic procedures. In retrospect it is amazing what the introduction of such a small aid, as the intratracheal tube, did to thoracic surgery. We look for perfection of such an aid to allow control of the circulation.

Mitral insufficiency has been treated by a number of people with few permanently good results. Bottles and baffles, sutures and slings, and prostheses of various types have failed to live up to their proponents' hopes. One of the most hopeful techniques at present is the suture constriction of the atrioventricular ring. This constricts the valve ring and makes what tissue remains more effective in closing the smaller hole. Certainly, to treat the loss of tissue which occurs from scar contraction and the dilatation of the valvular ring in an area so vitally important as the mitral, one requires direct vision and improvements in our techniques of suture and valve replacement. This is a common lesion and one of the major challenges to cardiovascular investigators.

Aortic stenosis has been treated with essentially the same principle as mitral stenosis, namely, an attempt to split the valvular commissures. Unfortunately the aortic valve becomes much more densely involved, almost always calcified, and almost never can a functionally normal valve be obtained. Untreated patients with aortic stenosis do well for many years and then fail rapidly once failure begins. Probably for this reason operation is not indicated until the later stages are reached and then the procedure is associated with a 10 to 20 per cent mortality.

The treatment of aortic insufficiency requires replacement of lost tissue and this also is far from a solved problem. The best solution to date is that supplied by Hufnagel who inserts a lucite ball valve in the upper thoracic aorta. His results have been good in about half his patients; about a fourth have died early following operation, and another fourth in the later postoperative period. It must be added, however, that he had as many patients die in the hospital during study and awaiting operation as in the early postoperative period and that operation upon healthier patients undoubtedly would be associated with better results. This valve, like the mitral, presents difficult problems of the loss of vital substance in a critical and high pressure area. The origin of the coronary arteries near by adds to the surgeon's troubles.

As is known, and as I have tried to summarize, recent progress in this field has been phenomenal. Should we continue to make such strides these and many other problems may be solved.

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PATHOLOGIC ASPECTS OF MITRAL STENOSIS

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APPRECIATION of the structural alterations of the mitral valve leading to rheumatic mitral stenosis depends in part upon understanding of certain anatomic features of the normal mitral valve. The normal mitral valve possesses two leaflets, a larger anterior and a smaller posterior. Between the two leaflets is connecting valvular tissue known as commissural tissue. One commissure lies in an anterolateral position, the other in a posteromedial. The two papillary muscles correspond in position with the commissures. There are, therefore, anterolateral and posteromedial papillary muscles. From each papillary muscle, chordae extend upward in a fan shape to the underlying valvular tissue. The center of the fan of chordae corresponds in position to that of the commissure. It is obvious that from each papillary muscle chordae extend to each leaflet. Stated otherwise, each leaflet has chordal connections with each papillary muscle. Each leaflet therefore has inserted into it the opposing groups of chordae.

In the development of rheumatic mitral stenosis, changes take place in the leaflets, in the commissures and in the chordae. The leaflets are thickened to varying degrees, the predominant amount of thickening being along the line of closure, corresponding to the location of the vegetations during the stage of active rheumatic endocarditis. It has been emphasized that in the usual instance the thickening and rigidity which develop in the altered mitral leaflet spare the basal attachment of the leaflet, leaving this area flexible. Varying amounts of calcified material may be deposited in leaflets altered by rheumatic fever.

Striking changes occur at the commissures. One change may be thickening of pre-existing commissural tissue with fibrous tissue alone or in association with calcified material. More striking than thickening of the commissure is elongation of it. This results from fusion of the two leaflets at the commissure.

The chordae lose their delicate threadlike quality, and in addition to thickening and shortening of the individual chordae there is fusion between them. This tends to obliterate the spaces which are normally present between the chordae. Shortening of the chordae tends to fix the mitral leaflets in a vertical position somewhat in line with the position of the normal opened mitral valve. Downward fixation of the leaflets coupled with elongation of the commissures by virtue of interadherence between the leaflets results in conversion of the two leaflets and associated chordae into a funnel-shaped structure, the apex of which is narrowest and lies at the ventricular level. The base of the funnel presents at the atrium. While the funnel-shaped deformity represents the most common picture in mitral stenosis, there may at times be a diaphragmlike deformity to the stenotic mitral valve. This is brought about by fusion between the two leaflets without the associated significant chordal involvement. The chordae are, therefore, of essentially normal length and do not cause downward deformity of the leaflets. The leaflets are fused in the position comparable to the closed position of the normal mitral valve.

There is an exceptional instance wherein only minor changes occur in the chordae and in the commissures but in which stenosis is caused by excessive rigidity, usually with calcification of the valve leaflets.

There are two common beliefs which are not necessarily true. One is that dense calcification of the mitral valve signifies mitral stenosis rather than insufficiency and the other is that when mitral stenosis exists mitral insufficiency is always an accompanying feature. While mitral insuffi-

ciency may coexist with mitral stenosis, in the predominant number of cases of mitral stenosis there is no significant associated insufficiency.

As one examines the mitral valve of the funnel type of stenosis it is apparent that in addition to the basic valvular and chordal changes leading to the funnel type of stenosis, there is a characteristic deformity of the anterior leaflet. This deformity consists of a convexity of the anterior leaflet, which convexity is directed toward the posterior leaflet and upward into the atrium. In passing it may be pointed out that this deformity of the anterior leaflet creates a corresponding deformity of the outflow tract of the left ventricle and may be the feature responsible for the commonly present systolic murmur in pure mitral stenosis. As the left ventricle contracts, the anterior leaflet is pushed upward and in this way the crest of the deformity is brought into contact with the base of the posterior leaflet. Thus, while the apex of the funnel remains in a fixed state throughout the cardiac cycle the edges of the base of the funnel come in contact during ventricular systole preventing regurgitation. This feature may be stated as one in which the mitral valve is converted into a flutter type of valve when it is involved by the funnel type of stenosis.

As implied earlier, calcification may be responsible for mitral insufficiency by making the valves rigid throughout. Especially at the commissure the calcification maintains the two leaflets in a fixed position preventing apposition of the two. In such cases, while stenosis must be said to exist in a pathologic sense, the orifice may be of sufficient width to cause no significant disturbances if there is no insufficiency resulting from the rigidity caused by calcium deposits.

As regards mitral insufficiency, it is seen that one pathologic type may be the result of extensive calcification of the valve especially at the commissure. A second type of mitral insufficiency results from changes involving the posterior leaflet. These changes result in an over-all shortening of this leaflet. Part of this process comes from the scarring and retraction of the leaflet incident to the rheumatic process and part of it comes from posterior displacement of the mitral-valve ring. The processes leading to the posterior displacement of the mitral-valve ring are not exactly understood but appear to be related to dilatation of the left atrium. By study of the normal heart it is evident that the endocardium of the left atrium, the mitral ring and the posterior mitral leaflet are essentially a continuous structure so that tension on one element results in tension on the others. Thus, if the left atrium becomes dilated the ring is dislocated backward and with it the posterior leaflet is pulled away from the valvular orifice. If this process is sufficiently severe and especially if it is associated with intrinsic changes in the leaflet, mitral insufficiency may result from the failure of the two leaflets to come in contact during ventricular systole.

The contribution of the radiologist to the clinical diagnosis of mitral valvular disease is made to a large extent by prediction as to the size of the left atrium. It is superfluous to discuss here the subject of alterations in the position and contour of the esophagus by the enlarged left atrium. It may be of interest to present the changes in the tracheobronchial tree that result from the enlarged left atrium. As the left atrium enlarges, its peculiar position with regard to the tracheal bifurcation results in an increased angulation between the two major bronchi. This increased angulation appears largely to result from the effects upon the left main bronchus. This is displaced upward giving rise to an increase in the angle between the two bronchi as compared with the normal. Another phenomenon resulting from the upward enlargement of the left atrium and related to increase in angulation between the two bronchi is compression of the left main bronchus. The peculiar location and relation of the left main bronchus are pertinent to mention as the basis for understanding the factors leading

to compression of it. The left atrium lies below. The left main pulmonary artery ascends in front of the left main bronchus and proceeds to a level above this, coursing behind the branch of the left bronchus which goes to the upper lobe. In arching over the left bronchus the left pulmonary artery, therefore, tends to prevent the bronchus from rising while the enlarged left atrium exerts forces in the opposite direction. This results in narrowing of the lumen of the bronchus associated with the formation of a sharp rather than a rounded posterior edge.

Studies regarding the effects of the enlarged left atrium upon the tracheobronchial tree are under way in our laboratory. The features described represent preliminary observations on this subject.

One other subject remains to be discussed in regard to mitral stenosis and that is the anatomic effects on the pulmonary vascular tree. In essence, the elevated pulmonary capillary pressure which is part of the functional disturbance in mitral stenosis as well as in mitral insufficiency may be associated with structural changes in the pulmonary vascular tree. These take two forms. One is characterized by hypertrophy of the muscular layers of the small arteries and the other, and possibly a later phenomenon, is characterized by intimal fibrous thickening. The degree of luminal narrowing which may be caused by muscular hypertrophy is moderate and that by intimal fibrous proliferation may be severe. These structural changes have given some concern as to whether they might represent a barrier to restoration of good cardiac function following an adequately performed operation on the mitral valve. The indications are that these changes, however interesting, do not constitute impediments that are significant enough to affect the patient if an adequate valvulotomy is performed.

Mayo Clinic
Rochester, Minnesota

VOL. 76, NO. 4

CONGENITAL MITRAL STENOSIS

ROENTGEN STUDY OF ITS MANIFESTATIONS

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THE literature dealing with congenital mitral stenosis is rather sparse, which suggests the rarity of this condition. Of the 1,000 cases of congenital heart disease collected by Abbott,[1] isolated congenital mitral stenosis was found only 6 times. However, it was present accompanying some other defect such as a patent ductus arteriosus or interventricular septal defect in 17 other instances. Donnally,[7] in 1923, upon review of the literature was able to find only 11 cases of isolated congenital mitral stenosis and these cases were recognized only at autopsy.

Congenital mitral stenosis is not specifically mentioned in Taussig's[21] book entitled "Congenital Malformations of the Heart," published in 1947, further signifying the uncommonness of this anomaly. Furthermore, in a review of 22,000 consecutive autopsies recorded at the Johns Hopkins Hospital, only 2 clear cut instances of congenital mitral stenosis were found and both were complicated by other lesions. Also Jacobson, *et al.*[12] in reporting a case of congenital mitral stenosis associated with coarctation of the aorta indicate that this is the first recorded instance of this combination of defects in the literature. Consequently, it would appear that isolated congenital mitral stenosis is quite rare, and as a lesion complicating some other malformation is decidedly uncommon.

PATHOLOGIC ANATOMY AND PHYSIOLOGY

There is a decided difference of opinion as to the etiology of congenital mitral stenosis. Numerous authors[2,14,18,19,22] have stated with considerable assurance that congenital mitral stenosis is due to a fetal endocarditis. On the other hand, Gross,[11] as well as others[6,8,9,13,20] have been equally firm in their belief that congenital mitral stenosis is developmental in origin. Farber and Hubbard[10] have taken an intermediate position and they state there are two types of congenital mitral stenosis, one developmental due to hereditary deficiencies and the other secondary to fetal endocarditis.

Insufficient material is at hand to permit a detailed study of the stenotic mitral valve and such is beyond the province of this paper. In one of the cases from the Johns Hopkins Hospital autopsy series described by Oppenheimer,[17] there was thickening of the endocardium within the left ventricle and atrium (endocardial fibroelastosis) associated with congenital mitral stenosis. This specimen was obtained following autopsy of a white female, aged sixteen months, who was admitted to Johns Hopkins Hospital in critical condition. As shown in Figure 1, there was fusion of the mitral cusps at the commissures plus a partial fusion of the chordae tendinae with resultant shortening of these structures. There was also a coarctation of the aorta just distal to the origin of the left subclavian artery.

It is probable that some degree of insufficiency accompanies many cases of congenital mitral stenosis, and in this respect the congenital lesion is quite similar to acquired mitral stenosis. In several cases described later in this paper, there was undoubtedly some degree of mitral insuffi-

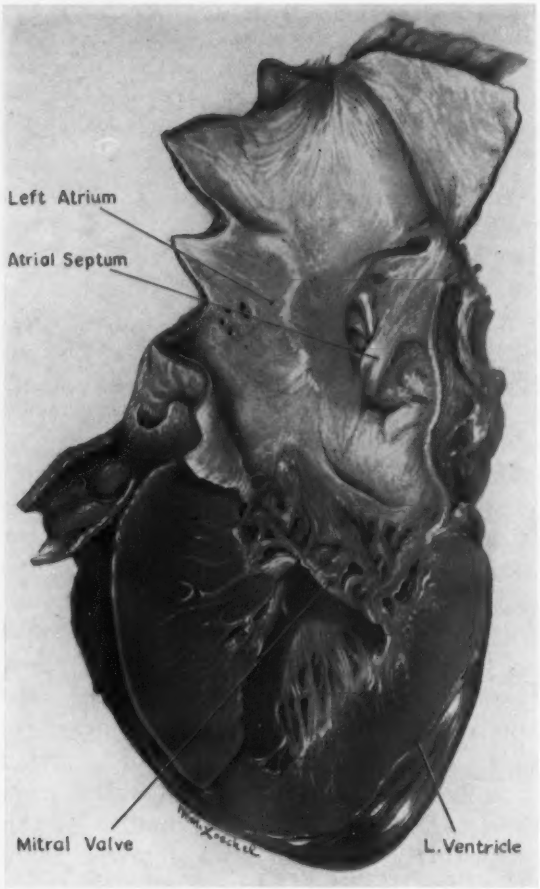

Fig. 1. Congenital mitral stenosis associated with endocardial fibroelastosis. Fusion of mitral cusps at commissures with partial fusion of chordae tendinae.

ciency, although the evidence would indicate that stenosis was the predominant abnormality.

The hemodynamic effects of congenital mitral stenosis are no different from those found in the acquired condition, although these effects tend to become manifest at an earlier age. In the uncomplicated case, there is an increased mean pressure in the left atrium, pulmonary veins, capillaries, arterioles, pulmonary artery and eventually in the right ventricle and atrium. Anatomically, this is manifest by dilatation of the left atrium and in some instances by hypertrophy of this chamber. There may also be some dilatation of the lung vascular structures accompanied, at times, by chronic passive congestion and intermittent attacks of pulmonary edema. Eventually as the hypertrophy and dilatation of the atrium occur there may be congestive heart failure. Marked or aneurysmal dilatation of the left atrium, comparable with that seen in acquired stenosis, is seldom observed in association with the congenital lesion, although the atrium may enlarge sufficiently to become border forming on the right side of the heart. This lack of great dilatation may be due to the relatively short duration of the disease when first observed, but it is more likely due to the fact that the atrial wall remains healthy and relatively strong, whereas in acquired stenosis due to rheumatic fever the atrial wall is often involved in the rheumatic process.

The natural course of uncomplicated congenital mitral stenosis would depend predominantly on the severity of the stenosis. It would seem that this condition would be similar in its effect to acquired mitral stenosis in which the rheumatic process is no longer active. In such instances a moderate degree of stenosis is compatible with longevity and only slightly restricted activity.

However, when congenital mitral stenosis is associated with another major anomaly, the disability due to these combined defects may be early in onset and severe. Mitral stenosis complicating a coarctation of the aorta may bring on early heart failure, and attempts at repair of the coarctation would be more hazardous and less likely to succeed. Furthermore an association of congenital mitral stenosis with patent ductus arteriosus may be one cause of a distinctive clinical syndrome, the chief feature of which is an uneven distribution of cyanosis between the upper and lower portions of the body.

MATERIAL AND PROCEDURES

The present report is based on the study of 2 proved and 15 presumptive cases of congenital mitral stenosis. In the 2 proved cases, the diagnosis was confirmed by autopsy, thus the associated defects were carefully studied. Fourteen of the 15 other

cases had definite evidence of left atrial enlargement by conventional roentgen examination. All 15 underwent angiocardiography, and in each instance there was stagnation of the contrast material within the left atrium. Furthermore, in 10 of the 15 cases, cardiac catheterization was carried out, and with one exception the pressure in the pulmonary artery was elevated. In 1 patient dilatation of the left atrium was confirmed at operation for coarctation of the aorta.

Admittedly, in those cases which did not come to autopsy, the diagnosis of mitral stenosis is not completely substantiated since there are other common causes of left atrial enlargement such as left ventricular failure and patent ductus arteriosus. In several of the cases of coarctation of the aorta with hypertension the possibility of left ventricular failure could be seriously considered. However, the angiocardiographic evidence of stagnation of contrast substance within the atrium suggests that the obstruction was due primarily to organic mitral stenosis rather than to relative mitral insufficiency secondary to left ventricular failure.

Rheumatic fever may be followed by mitral stenosis, but structural changes in the mitral valve due to this cause are rare during infancy and early childhood. In none of our patients was there a history of rheumatic fever.

For clarity in presentation, the cases of congenital mitral stenosis have been divided into groups according to the nature of the associated anomaly or defect.

GROUP I

MITRAL STENOSIS ASSOCIATED WITH A REVERSE FLOW THROUGH A PATENT DUCTUS ARTERIOSUS (3 CASES)

Case 1. E. R. was an eleven month old white female, admitted to the hospital with dyspnea and a history of cyanosis of both feet of three months' duration.

Physical Examination. There was clubbing of the toes and cyanosis of both feet and to a lesser extent of the left hand. The right hand, head and neck were normal in color. Blood pressure could not be obtained in any of the extremities, and the femoral pulsations were felt to be weaker than the brachial pulsations. There was a loud systolic murmur heard over the precordium but a definite diastolic murmur was not identified.

Roentgen Examination. Fluoroscopic examination with barium swallow demonstrated evidence of a large left atrium. Angiocardiographic study (Fig. 2*A*) shows that the contrast substance entered the heart in a normal manner and was expelled from the right ventricle through the pulmonary artery into the descending aorta. The communication between the pulmonary artery and the descending aorta was so large that the descending aorta appears as a continuation of the pulmonary artery. This is well demonstrated (Fig. 2*B*) in the lateral view showing the direct communication from the pulmonary artery into the descending aorta. Two days later a retrograde aortogram using the right brachial artery demonstrates the arch of the aorta (Fig. 2*C*) to a point at which the patent ductus arteriosus entered. Very little contrast substance was demonstrated beyond the point at which the ductus entered the aorta. This appearance was interpreted as being due to an obstruction or coarctation of the aorta at the usual site, and it was felt that the reverse flow through the patent ductus was the result of low pressure in the aorta beyond the coarcted area.

This patient was operated upon by Dr. Alfred Blalock. No obstruction or coarctation of the aorta could be found. During the course of the exploration, the patent ductus was ligated. The patient's heart promptly stopped beating and the patient could not be revived. At autopsy, there was thickening and stiffening of both cusps of the mitral valve with associated thickening and partial fusion of the attached chordae tendinae. The changes produced a significant stenosis of the valve. The left atrium was moderately dilated. A large patent ductus arteriosus opened into the aorta at the usual site. The left ventricle was atrophic and there was narrowing of the aortic ring producing a mild degree of aortic stenosis. In addition, the ascending aorta was considerably diminished in caliber.

Comment. Evidently there was a large minute volume of blood shunted from the pulmonary artery into the descending aorta, causing a marked dilution of the con-

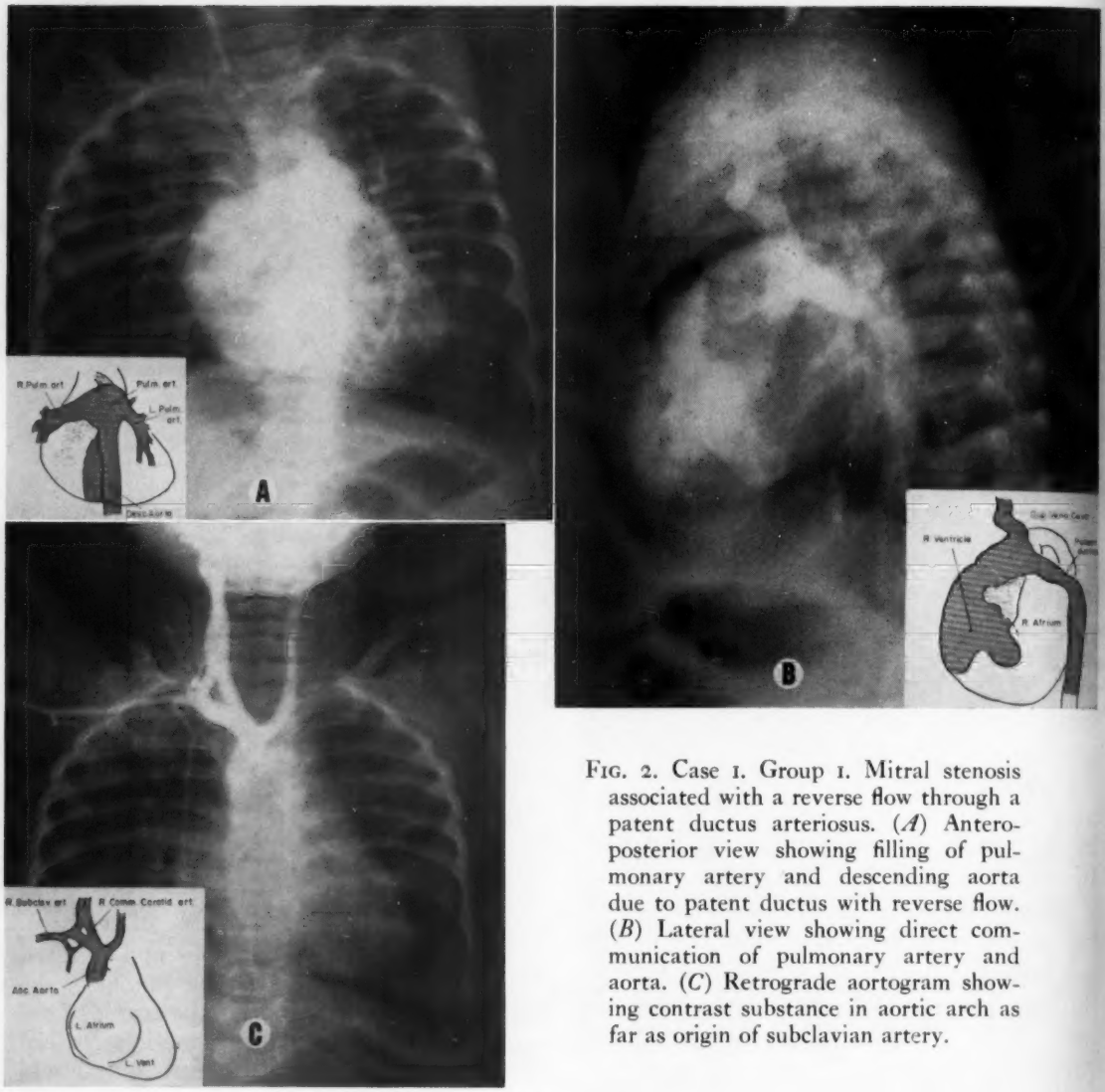

Fig. 2. Case I. Group I. Mitral stenosis associated with a reverse flow through a patent ductus arteriosus. (*A*) Anteroposterior view showing filling of pulmonary artery and descending aorta due to patent ductus with reverse flow. (*B*) Lateral view showing direct communication of pulmonary artery and aorta. (*C*) Retrograde aortogram showing contrast substance in aortic arch as far as origin of subclavian artery.

trast substance in the aorta at the point of entrance of the ductus at the time of aortography. This led to the erroneous diagnosis of coarctation of the aorta. An insufficient amount of the contrast material reached the left atrium to allow visualization and hence the mitral stenosis was unsuspected. However, because of the experience obtained in this case we have been able to strongly suspect a similar condition in 2 additional cases following angiocardiography.

Case II. A. L., a white female fourteen months of age was apparently healthy at birth but at ten days of age cyanosis of the hands and feet was noticed when the child cried. A heart murmur was heard at two weeks of age and paroxysmal dyspnea appeared shortly thereafter.

Physical Examination. There was cyanosis of both feet and to a lesser degree of the left hand. The right hand and head were normal in color. The pulmonary second sound was loud and split. A systolic murmur was audible over the precordium but no diastolic murmur was heard following repeated examinations.

Roentgen Examination. Fluoroscopy and roentgenograms showed globular enlargement of the heart. The lung vascularity was normal.

There was posterior deviation of the barium-filled esophagus indicating enlargement of the left atrium. Also in the frontal projection (Fig. 3*A*) there was a rounded density superimposed on the base of the heart on the right side indicating left atrial enlargement. Angiocardiography (Fig. 3, *B* and *C*) showed that contrast substance passed in high concentration from the large pulmonary trunk directly into the descending aorta. About three seconds later (Fig. 3*D*) the left atrium became opaque and the contrast substance stagnated for a period of several sec-

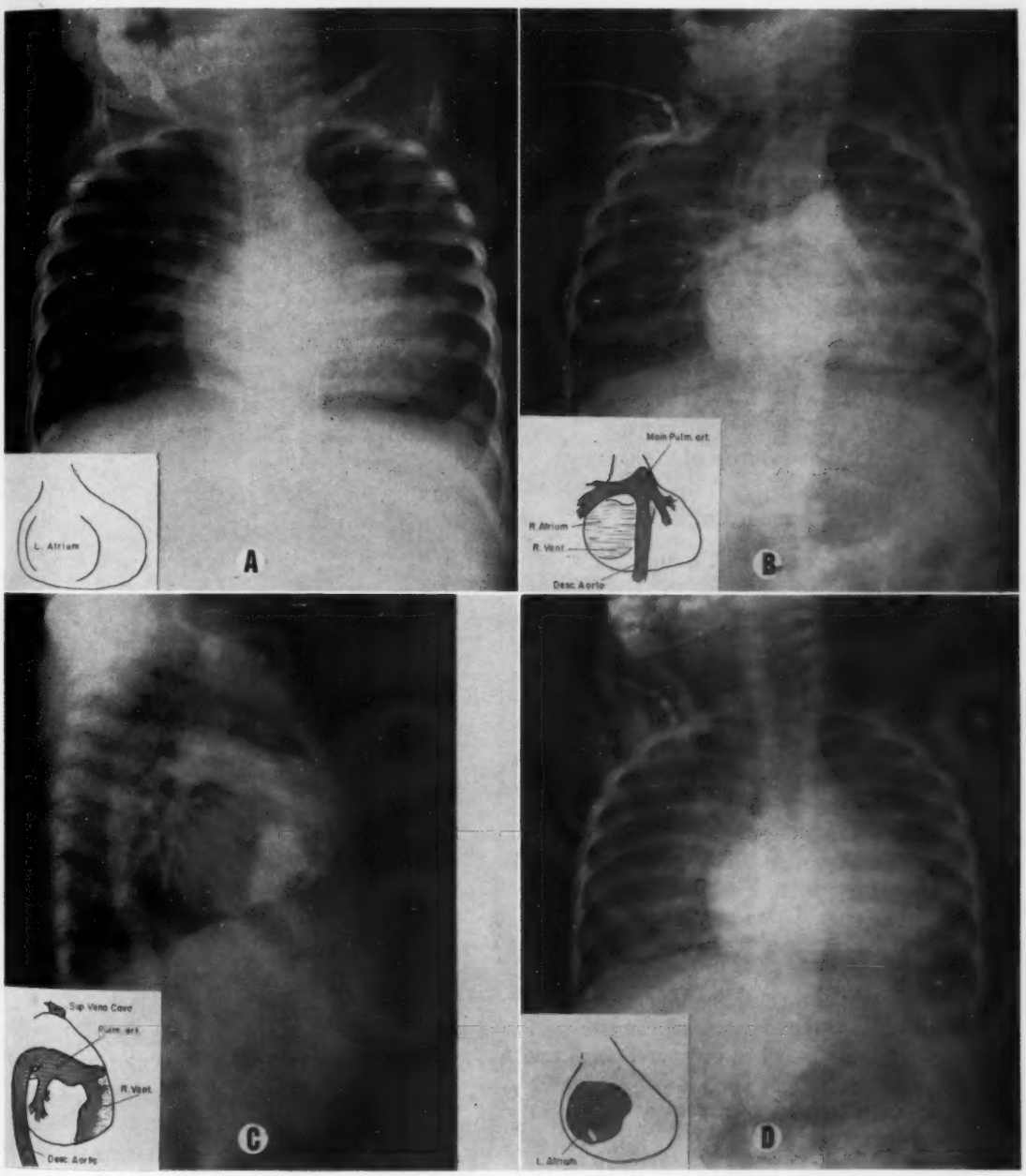

Fig. 3. Case 11. Group 1. Mitral stenosis associated with reverse flow through a patent ductus arteriosus. (*A*) Plain roentgenogram showing large left atrium. (*B*) Anteroposterior angiocardiogram, showing contrast medium in pulmonary artery and aorta. (*C*) Lateral view showing contrast substance passing in high concentration from pulmonary artery directly into descending aorta. (*D*) Delayed anteroposterior roentgenogram showing contrast substance stagnating in left atrium.

onds. The left ventricular cavity was not visualized.

Cardiac catheterization (performed by Dr. H. T. Bahnson) showed a right ventricular pressure of 134/28 and a pulmonary artery pressure of 99/0. The catheter was passed from the pulmonary artery through a patent ductus into the descending aorta.

The patient was discharged without further attempts at definitive treatment.

Comment. A reverse flow of blood through a patent ductus was unequivocally demonstrated. There was stagnation of the contrast material in a dilated left atrium presenting good evidence of obstruction at the mitral valve, thought to be due predominantly to a mitral stenosis. There is no evidence for or against the presence or aortic stenosis. This case is very similar to Case 1 in this Group and also the case reported by Azevedo, *et al.*[3] in which an antemortem diagnosis of congenital mitral stenosis with reverse flow through a patent ductus was made following angiocardiography.

Case III. (Case presented through the courtesy of Drs. J. E. Miller and Robert R. Shaw of Dallas, Texas.)

J. H., a seven and one-half year old white female was premature but otherwise was thought to be normal at birth. She developed poorly, however, and during the past two to three years there had been occasional spells of unconsciousness. Cyanosis of the face was noticeable following exercise and there was marked shortness of breath.

Physical Examination. There was definite orthopnea and dyspnea. The blood pressure in the arms was 95/60. There was a 3 plus systolic murmur and also a rumbling mid-diastolic murmur. The pulmonary second sound was loud and split. There was no clubbing of the extremities but careful inspection showed the toe nails to be slightly cyanotic when compared with the finger nails.

Cardiac catheterization revealed the right ventricular pressure to be 63/9 with an increased pressure in the pulmonary artery. A femoral artery blood sample showed a 10.9 volume per cent oxygen content and a saturation of 48 per cent.

Roentgen Examination. There was a large globular heart. The lung vascularity was within normal limits although there was evidence of dilatation of the pulmonary trunk. There was a wide posterior sweep of the barium filled esophagus (Fig. 4*A*) suggesting some dilatation of the left atrium.

Angiocardiography (performed by Dr. J. E. Miller) showed that contrast material (Fig. 4*B*) passed immediately from the right ventricle into a dilated pulmonary trunk and then into the descending aorta. Only a small amount of the contrast substance reached the peripheral pulmonary arteries. At the end of about four seconds a quite large dilated left atrium (Fig. 4*C*) was clearly outlined and remained opacified for several seconds. The left ventricle and ascending aorta were not opacified at any time during the examination.

The patient was discharged without any definitive treatment, She died about one year later but no autopsy was obtained.

Comment. It is possible that in addition to mitral valvular disease there was an aortic stenosis or a coarctation in this case although there is no direct evidence of either. This patient was somewhat older than the majority of patients seen in this series. The left atrium was evidently quite large and approached the size which is sometimes seen in acquired mitral valvular disease.

GROUP II

CONGENITAL MITRAL STENOSIS WITH A PATENT DUCTUS ARTERIOSUS WITHOUT A REVERSE FLOW (3 CASES)

In all three cases of this group there were low pitched mid or late diastolic murmurs there was also definite evidence of enlargement of the left atrium and an elevated pulmonary artery pressure. A patent ductus arteriosus with a later development of pulmonary hypertension can bring about the same combination of findings, although with such a combination the presence of a definite diastolic murmur would be unexplained. Likewise, the left atrium in one case (M. K.) was somewhat larger than that seen in uncomplicated patent ductus arteriosus.

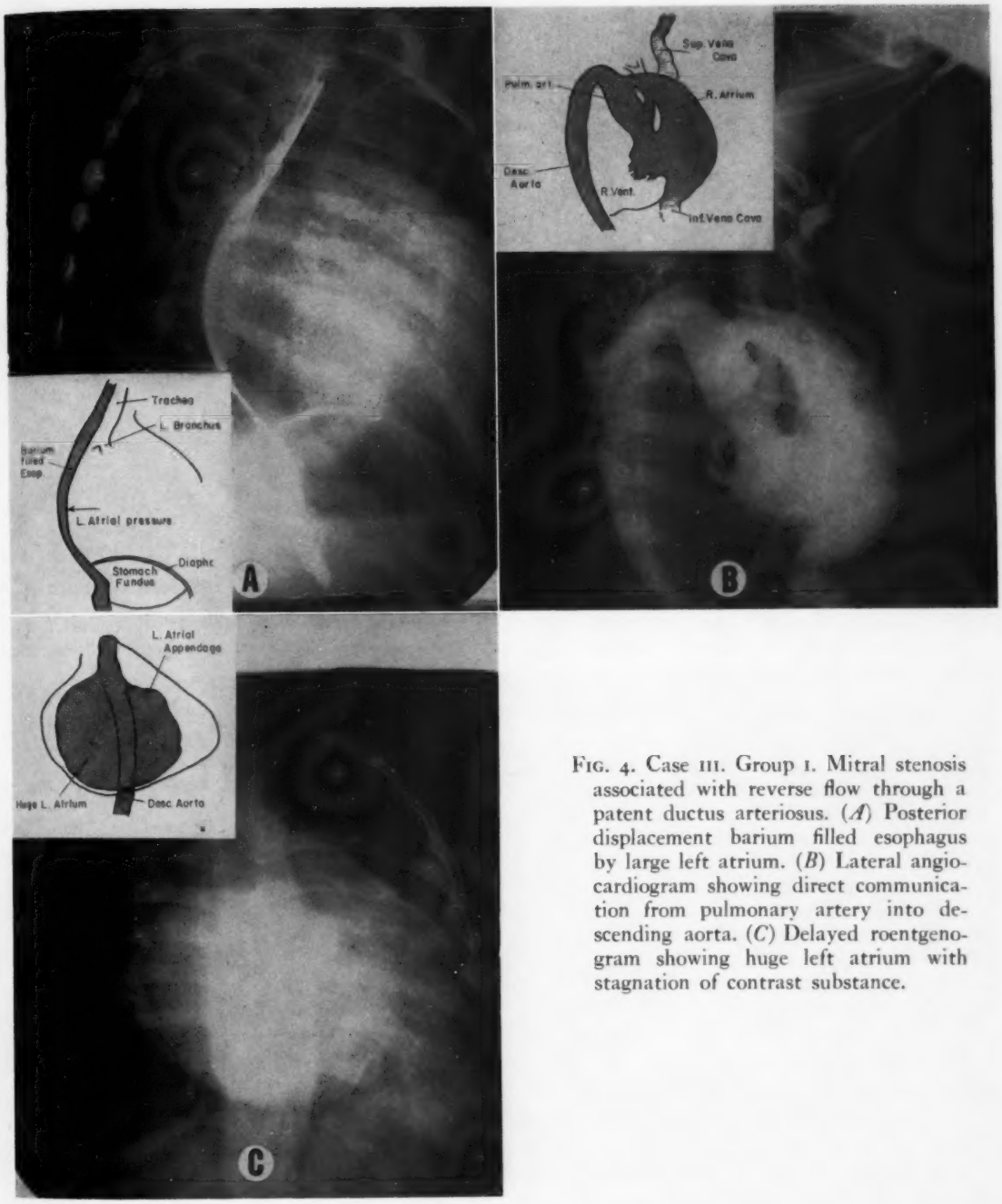

FIG. 4. Case III. Group I. Mitral stenosis associated with reverse flow through a patent ductus arteriosus. (*A*) Posterior displacement barium filled esophagus by large left atrium. (*B*) Lateral angiocardiogram showing direct communication from pulmonary artery into descending aorta. (*C*) Delayed roentgenogram showing huge left atrium with stagnation of contrast substance.

CASE I. T. M. was a four and one-half year old white male thought to be normal at birth, but a heart murmur was heard at one month. The patient breathed rapidly, squatted frequently and was often cyanotic following mild exertion.

Physical Examination. There was no clubbing or cyanosis. The heart was markedly enlarged. There was an apical systolic thrill and murmur and in addition there was a not too well localized diastolic murmur.

Cardiac catheterization demonstrated a pulmonary artery pressure of 71/48 and a wedge pressure taken from a distal pulmonary artery (equivalent to pulmonary capillary pressure) was also elevated. Also there was a 2 volume per

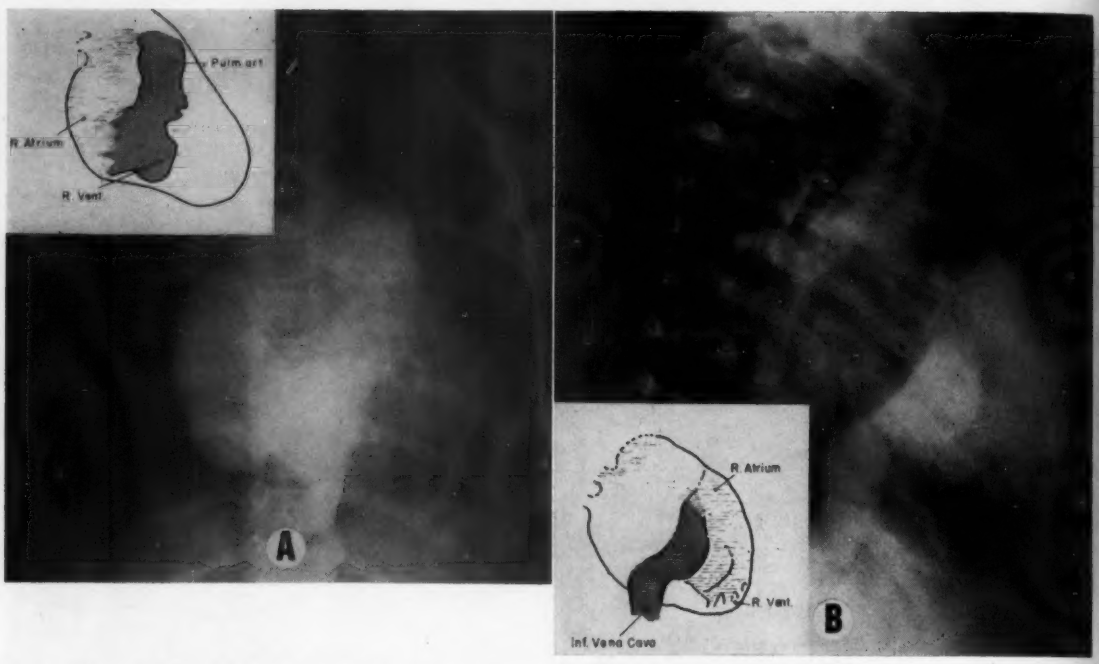

Fig. 5. Case I. Group II. Congenital mitral stenosis with patent ductus arteriosus without reverse flow. (*A*) Marked dilatation pulmonary artery. (*B*) Lateral view showing convexity posterior wall right atrium due to pressure from large left atrium.

cent increase in oxygen saturation in blood samples taken from the pulmonary artery and its branches as compared with those taken from the right ventricle.

Roentgen Examination. Fluoroscopy showed a marked globular cardiac enlargement with a dilated pulmonary artery and an increase in prominence of the pulmonary vascular markings. Angiocardiography (Fig. 5*A*) revealed a marked dilatation of the pulmonary artery and its branches. In the lateral view (Fig. 5*B*) there was a convexity of the posterior wall of the right atrium due to pressure from the greatly enlarged left atrium.

The patient was discharged as unimproved without attempts at definitive treatment.

Case II. M. K., a six year old white female who was thought to be normal at birth but at six weeks a heart murmur was noted. Patient became dyspneic on exertion and at nine months of age during a respiratory infection, cyanosis was observed but this quickly disappeared.

Physical Examination. The patient was quite thin and there was a bulge of the anterior chest wall. The blood pressure was 110/60. There was a loud systolic murmur at the apex and also a well localized low pitched mid diastolic murmur in the third left intercostal space.

Cardiac catheterization showed a pressure in the main pulmonary artery of 69/56 and an increase of 2 volume per cent in the blood samples taken from the pulmonary artery as compared with those from the right ventricle.

Roentgen Examination. The heart was greatly enlarged and the left atrium formed part of the right upper heart border. There was an increase in caliber of the pulmonary vascular structures.

Angiocardiography (Fig. 6*A*) showed stagnation of the contrast substance in the greatly dilated left atrium (Fig. 6*B*) and a concavity in the posterior wall of the right atrium due to pressure from the large adjacent left atrium.

The patient was discharged as unimproved without any attempts at definitive treatment.

Case III. This case is not described in detail but was similar in most respects to the other 2 cases.

GROUP III

CONGENITAL MITRAL STENOSIS ASSOCIATED WITH COARCTATION OF THE AORTA (6 CASES)

In 1 case the diagnosis was confirmed by autopsy. In 5 other cases the clinical, laboratory and angiocardiographic findings were quite compatible with the diagnosis of mitral stenosis.

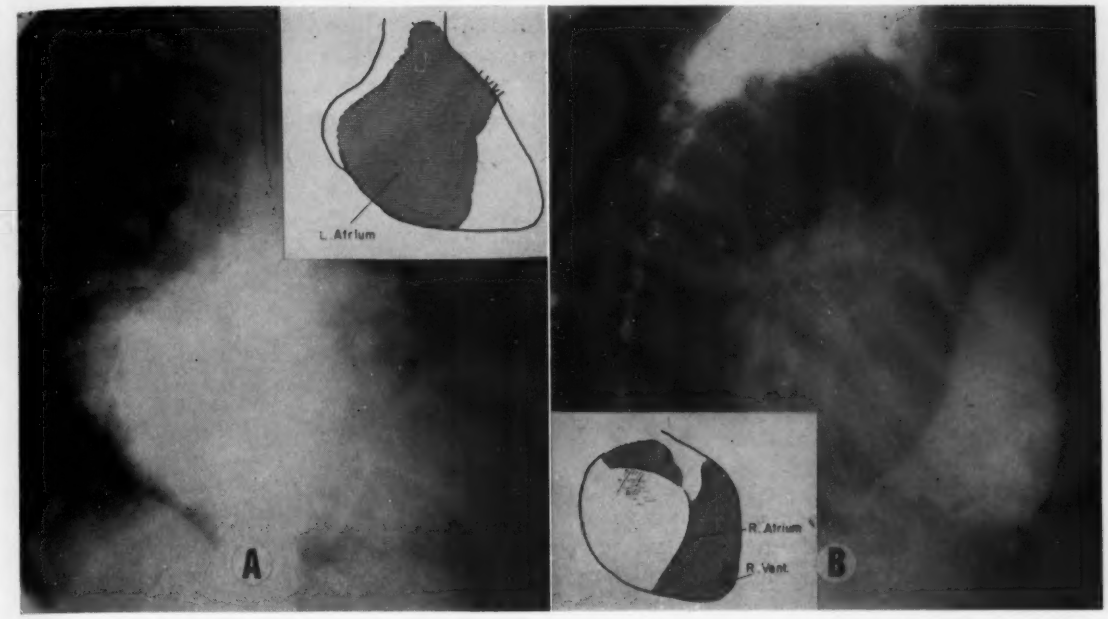

Fig. 6. Case II. Group II. Congenital mitral stenosis with patent ductus arteriosus without reverse flow. (*A*) Stagnation contrast substance left atrium. (*B*) Lateral view showing concavity posterior wall right atrium due to pressure large left atrium.

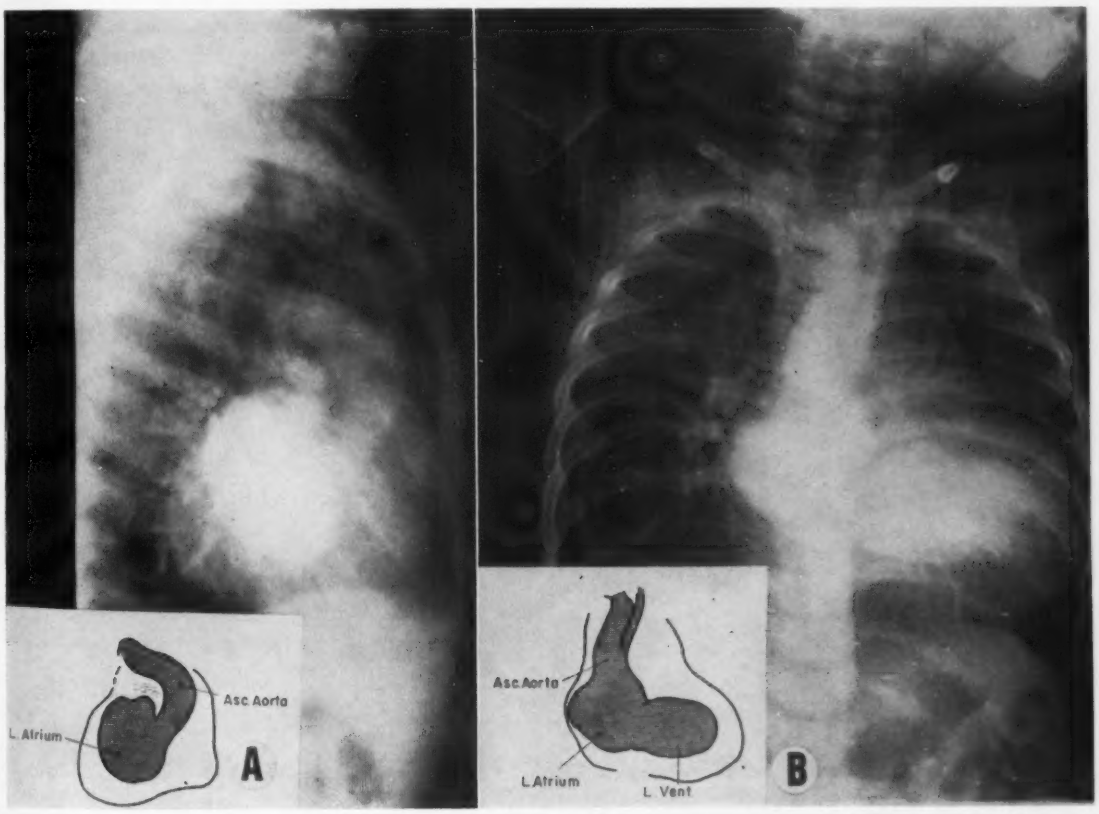

Fig. 7. Case I. Group III. Congenital mitral stenosis associated with coarctation of aorta. (*A*) Anteroposterior view showing left atrial and ventricular enlargement. (*B*) Lateral view demonstrating large left atrium and coarctation of aorta.

Case I. D. D. was a ten month old white female who was thought to be normal at birth. She had been essentially asymptomatic except for an attack of rapid breathing. A heart murmur was heard and there was a mild deformity of the left side of the thorax; hence the patient was referred to the hospital for study.

Physical Examination. There was a prominence of the left lateral thoracic cage. A short early systolic murmur and a systolic thrill were heard at the apex. Also there was a Grade 1 rumbling diastolic murmur to the left of the sternum. Blood pressure in the upper extremities was 160/100 and in the lower extremities 70/50. An electrocardiogram revealed evidence of left ventricular strain and atrial dilatation.

Roentgen Examination. The heart was slightly enlarged. An anteroposterior angiocardiogram demonstrated left atrial and ventricular enlargement (Fig. 7*A*) and a subsequent lateral film (Fig. 7*B*) revealed coarctation at the usual site.

Because of the probable presence of a mitral stenosis, the surgical treatment of the coarctation was deferred and the patient was sent home.

Comment. Some degree of left ventricular failure could have been present in this case and thus produced a degree of left atrial enlargement. However, there was a rumbling diastolic murmur which was suggestive of mitral stenosis.

Case II. K. G. was a white male, aged two and one half, who was admitted to Johns Hopkins Hospital with a history of tiring very easily, shortness of breath on exertion and cyanosis around the lips. At the age of three months a diagnosis of congenital heart disease had been made by the family physician.

Physical Examination. The blood pressure in the right arm was found to be 160/85. A blood pressure could not be obtained in the legs except by flush technique and it was obviously quite low. There was a faint thrill over the mitral area and a blowing Grade 2 mitral systolic murmur was heard. On physical examination the heart was found to be enlarged.

Roentgen Examination. Lateral angiocardiogram showed a large left auricle and, to a lesser extent, left ventricle, with contrast substance filling the aorta and presenting a rather typical coarctation at the usual site (Fig. 8*A*). An anteroposterior film again demonstrated a large ascending aorta and a large left auricle with a

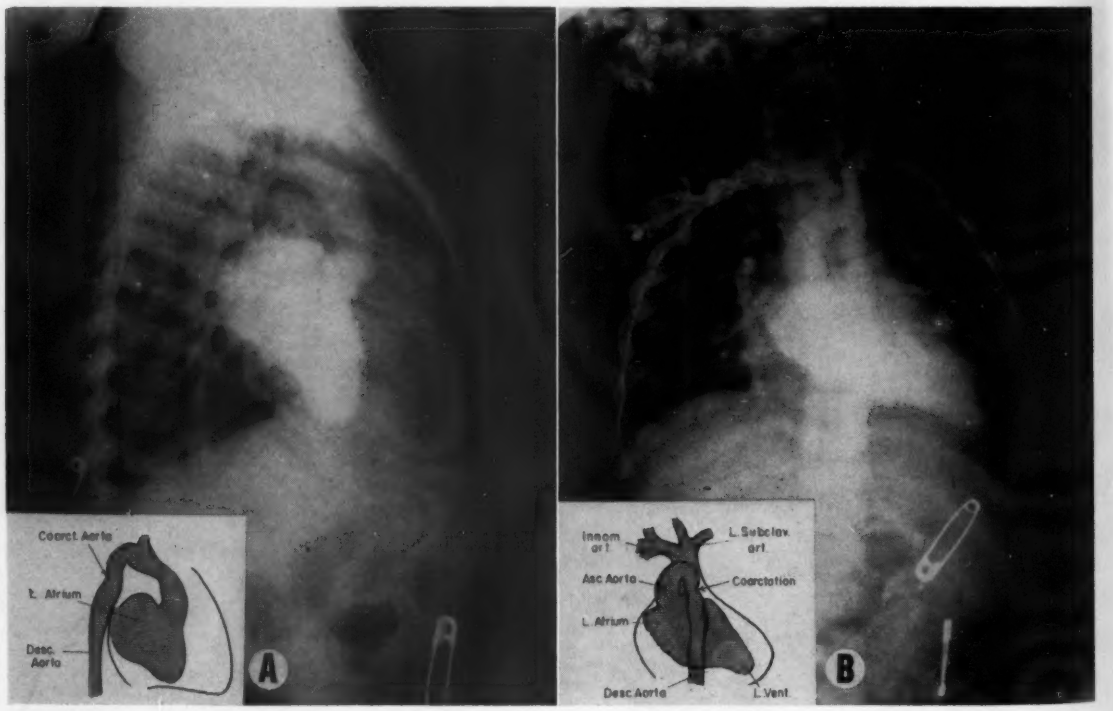

Fig. 8. Case III. Group III. Congenital mitral stenosis associated with coarctation of aorta. (*A*) Lateral view showing characteristic site of coarctation and large left atrium. (*B*) Large left atrium and typical coarctation of aorta.

typical coarctation of the aorta just distal to the origin of the left subclavian artery (Fig. 8*B*).

This patient was studied rather completely in the cardiac clinic and coarctation plus mitral valvular disease was strongly suspected. The patient was discharged to the follow-up clinic. Surgery was contemplated at a later date if there was sufficient indication.

There were four additional patients in this group, one of whom died. At autopsy (Fig. 1) a mitral stenosis with a coarctation of the aorta was verified.

GROUP IV

MITRAL STENOSIS WITH PROBABLE INTERVENTRICULAR SEPTAL DEFECT (2 CASES)

In these 2 cases the evidence for the presence of an interventricular septal defect was obtained by cardiac catheterization.

Case I. E. P. was a six year old white female who was apparently normal at birth. At the age of nine weeks a murmur was noted at the apex of the heart. The patient was retarded in development and had always been a feeding problem.

Physical Examination. The heart was enlarged and there was bulging of the anterior chest wall on the left. There was a rumbling mid diastolic murmur near the apex and also a loud systolic murmur.

Roentgen Examination. The heart was symmetrically enlarged. The pulmonary vascular markings were increased in prominence (Fig. 9*A*) and the superimposed shadow of the left atrium on the base of the heart could be identified in the plain roentgenogram. Angiocardiography (Fig. 9*B*) showed a concavity of the posterior wall of the right atrium due to the pressure from the adjacent left atrium; also (Fig. 9*C*) there was stagnation in the left atrium. Subsequent roentgenograms demonstrated some dilution of the contrast substance in the pulmonary artery.

Cardiac catheterization revealed a right ventricular hypertension with an increase in oxygen content in blood taken from 9.4 in the right auricle to 10.4 volume per cent in the right ventricle.

Based on clinical, laboratory, and angiocardiographic studies, a diagnosis of congenital mitral stenosis associated with an interventricular septal defect was made.

There was 1 additional case in this group with similar findings.

GROUP V

UNCOMPLICATED CONGENITAL MITRAL STENOSIS (3 CASES)

Case I. R. S. was a white male of three and one-half years of age who had a cardiac murmur at birth but otherwise was quite normal. The patient had no symptoms except that his heart seemed to beat more rapidly than normal. There was no history of rheumatic fever.

Physical Examination. The heart was not enlarged. There was a rumbling low pitched diastolic murmur with a thrill at the apex.

Cardiac Catheterization. At rest the pressure in the right ventricle and pulmonary artery was normal but following exercise there was a definite elevation of pressure, indicating some obstruction to blood flow beyond the pulmonary artery which was thought to be due to a mitral stenosis.

Roentgen Examination. The heart was not enlarged and its contour was within normal limits (Fig. 10*A*). However, angiocardiography (Fig. 10*B*) showed stagnation of the contrast material in a dilated left atrium. There was also some dilatation of the pulmonary artery.

The patient was discharged as unimproved but has been able to live a normal or only slightly restricted life at home.

Comment. This represents apparently a mild case of mitral obstruction and whether it will increase in severity remains to be seen.

Case II. D. M. was a white female, fourteen months of age, who was admitted to the hospital suffering from congestive heart failure.

Physical Examination. There was an apical systolic murmur and a gallop rhythm. There was no cyanosis or clubbing of the extemities.

Cardiac Catheterization. The pulmonary artery pressure was 78/38.

Roentgen Examination. Fluoroscopy and roentgenograms showed a large globular heart with evidence of left atrial enlargement. Angiocardiography (Fig. 11, *A* and *B*) showed dilatation of the pulmonary artery and stagnation of the contrast material in a dilated left atrium.

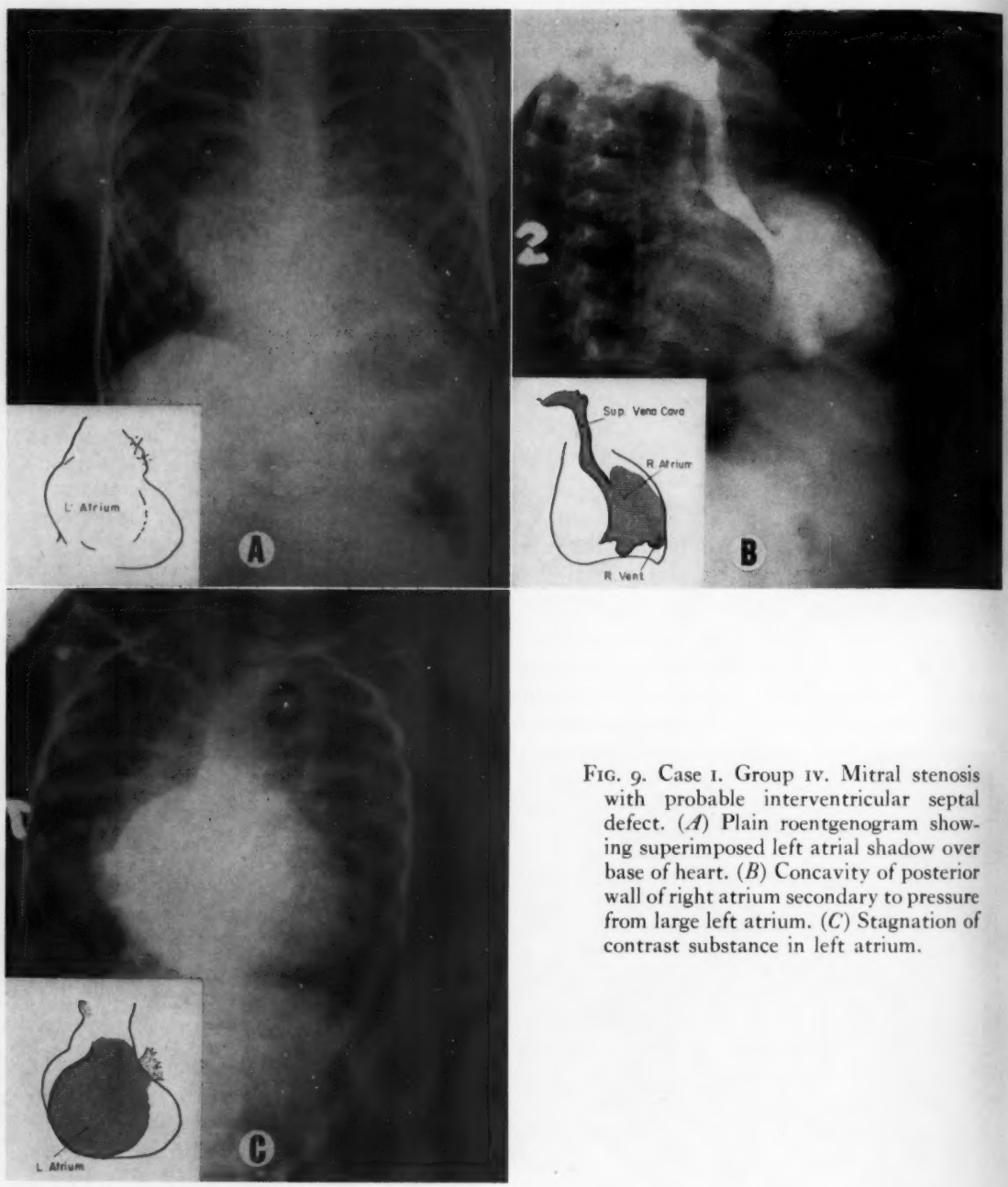

FIG. 9. Case 1. Group IV. Mitral stenosis with probable interventricular septal defect. (*A*) Plain roentgenogram showing superimposed left atrial shadow over base of heart. (*B*) Concavity of posterior wall of right atrium secondary to pressure from large left atrium. (*C*) Stagnation of contrast substance in left atrium.

An exploratory thoracotomy demonstrated a large pulmonary artery and a thick walled right ventricle. The aorta was smaller than normal. No definitive surgical treatment was attempted. From the anatomic findings the surgeon thought that a mitral stenosis or atresia was present. The patient was discharged as unimproved.

DISCUSSION

Dammann, *et al.*[4] have described a syndrome of "Patency of the ductus arteriosus with pulmonary hypertension and a shunting of blood from the pulmonary artery to the aorta." One of the features of this syndrome is an increased cyanosis of

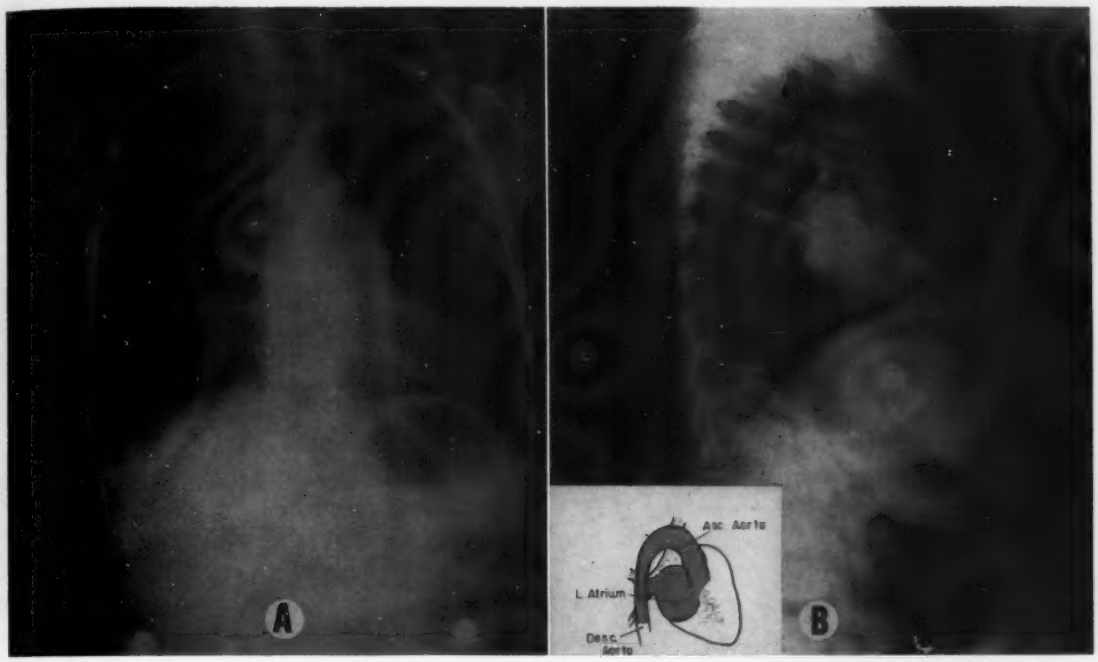

Fig. 10. Case 1. Group v. Uncomplicated congenital mitral stenosis. (*A*) Plain roentgenogram showing heart which is normal in size and configuration. (*B*) Lateral angiocardiogram showing stagnation of contrast medium in dilated left atrium.

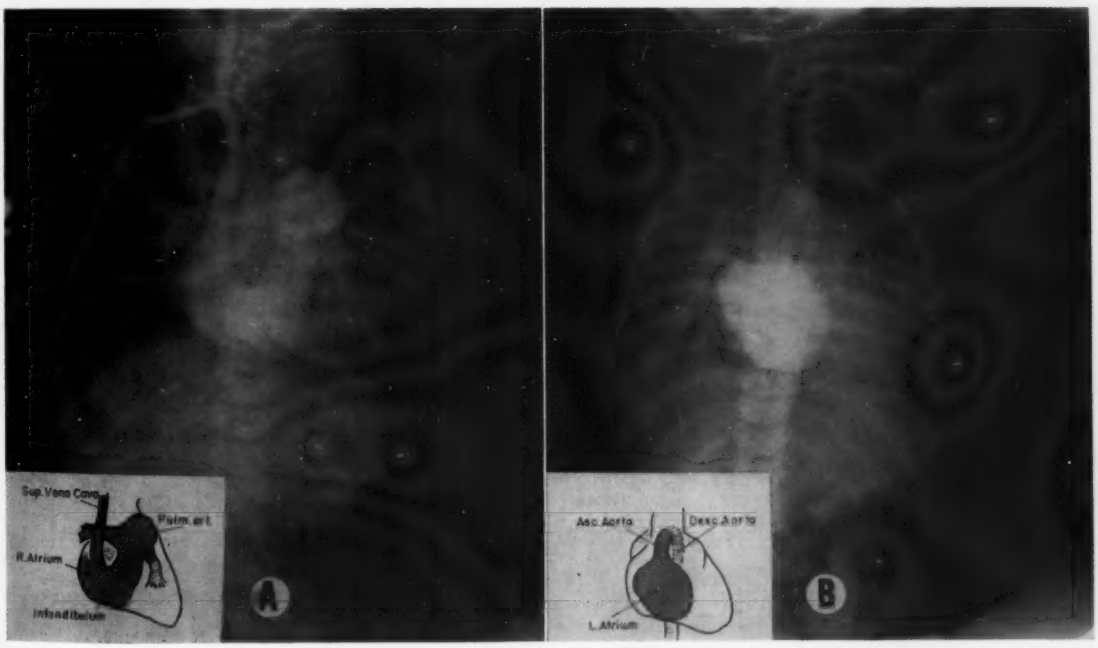

Fig. 11. Case 11. Group v. Uncomplicated congenital mitral stenosis. (*A* and *B*) Anteroposterior views following angiocardiography demonstrating dilatation of pulmonary artery and stagnation of contrast medium in dilated left atrium.

the lower extremities as compared with the upper extremities and head. The authors attributed the pulmonary hypertension in the cases which they studied at autopsy to narrowing of the pulmonary vascular bed due to multiple small thrombotic areas in the small pulmonary arteries and arterioles. It now appears that congenital mitral stenosis may be the cause of a similar syndrome and such a condition has already been diagnosed antemortem by Azevedo, *et al.*[3] In Case 1, Group 1 of our series the syndrome was clearly present although the mitral stenosis was unsuspected before death. In the 2 other cases the diagnosis is yet unconfirmed.

One can only speculate about the effects of a congenital mitral stenosis upon the closure of a patent ductus. An early reversal of flow through the ductus might conceivably serve to maintain its patency.

A reversal of flow through a patent ductus arteriosus may occur in association with any combination of circumstances which brings about a higher mean pressure in the pulmonary artery than in the thoracic aorta. Dammann and Sell[5] have reported a number of instances of pulmonary hypertension in association with patent ductus arteriosus and similar observations have been made by Myers, *et al.*[15] In the 2 cases described by Myers, *et al.* there was definite enlargement of the left atrium although there was no evidence of reversal of flow or cyanosis. It is well known that dilatation of the left atrium is a frequent occurrence in association with uncomplicated patent ductus arteriosus and probably with those cases which develop a significant pulmonary hypertension. Angiocardiography should be helpful in differentiating between a patent ductus arteriosus and the same condition complicated by a mitral stenosis.

One case of congenital mitral stenosis associated with coarctation of the aorta was found by Oppenheimer[17] in a series of 5 infants with extensive endocardial fibroelastosis. Olney and Stephens[16] also found unsuspected congenital mitral stenosis at autopsy in 2 of 5 children with coarctation of the aorta. Jacobson, *et al.*[12] report a similar condition in a two year old patient who died following an attempted resection of the coarctation. The present series suggests that coarctation and congenital mitral stenosis occur together more frequently than one would expect by chance association. Since the basic cause of each anomaly is unknown, the reason for this association is obscure. Certainly these observations should serve to make the cardiologist and surgeons aware of another possible complication of coarctation of the aorta particularly when seen in childhood.

Most of the patients with this double lesion were in severe distress and running a downhill course. The question of advisability of attempting a mitral valvulotomy or commissurotomy in conjunction with the resection of the coarctated aorta or at a later time has not been determined since the anatomy of congenital mitral stenosis has not been sufficiently studied so as to determine its susceptibility to the procedures used in acquired mitral stenosis.

SUMMARY

1. A total of 17 cases thought to represent instances of congenital mitral stenosis have been studied roentgenologically in this series. Two of these cases were proved by autopsy. In every instance in which angiocardiography was performed, there was evidence of stagnation of the contrast substance within the left atrium and in every case except one the left atrium was appreciably enlarged.

2. It appears that congenital mitral stenosis occurs more commonly in conjunction with some associated anomaly than as an isolated lesion. This is borne out by our present series of cases as well as by reports in the literature. Furthermore, recent evidence from our own experience as well as other case reports suggests that congenital mitral stenosis, while still rare, may be more common than previously considered.

3. Finally, it is believed that congenital mitral stenosis can and should be diagnosed

by modern diagnostic procedures prior to death. Hence these children may, in selected cases, be afforded an opportunity of a prolonged life by early and skillful cardiac surgery.

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BILATERAL DUCTUS ARTERIOSUS WITH RIGHT AORTIC ARCH AND RIGHT-SIDED DESCENDING AORTA

REPORT OF CASE

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IN THE field of malformations of the aortic-arch system the occurrence of bilateral ductus arteriosus is infrequently reported. Among the relatively few instances of this condition recorded in the literature, it has been common for associated serious intracardiac defects to be present.[1,4,8,10] In other instances, bilateral ductus arteriosus is associated with absence of the proximal portion of a pulmonary artery.[7]

In the case of bilateral ductus arteriosus that we are reporting, there were no major intracardiac malformations and the pulmonary arteries were normally developed. Wide patency of the right ductus arteriosus, however, may have accounted for the early death of the infant in this case.

The number of cases of right aortic arch with right-sided descending aorta reported in the recent literature has been sufficient to make this particular vascular configuration a generally recognized one.[2,3,5,6,11] In the usual instances, however, there is only one ductus arteriosus, a right or a left. In some patients with intracardiac defects both the right and the left ductus arteriosus may be absent. In the more common varieties of right aortic arch with right-sided descending aorta, the left subclavian artery arises either from a left-sided innominate artery in company with the left common carotid artery or as the fourth branch of the aorta and passes behind the esophagus to gain the left side of the body. As far as we are aware, except for Ghon's case reported in 1908 no recently reported example is very similar to the one we are presenting. In it a right aortic arch with right-sided descending aorta was associated with bilateral ductus arteriosus. The left subclavian artery had no connection with the aorta and arose from the left ductus arteriosus.

REPORT OF CASE

Clinical Features. A male infant, born on June 14, 1955, died at the age of six days. At the time of birth he had a hoarse cry; he nursed poorly. On the fourth day of life vomiting occurred. No heart murmurs were heard. The lungs appeared normal on examination. Roentgenographic examination of the thorax showed widening of the superior cardiac shadow with particular prominence on the right. The pulmonary hilar vessels were prominent (Fig. 1).

FIG. 1. Roentgenogram showing prominence of right hilar vessels and of right superior mediastinum.

On the fifth day cyanosis with grunting respiration developed; there was temporary improvement under treatment with oxygen, but gallop rhythm ensued and death occurred on the sixth day of life.

Pathologic Features. The site of chief interest was the great vessels. (Fig. 2 and 3). The aorta arose from the left ventricle in a normal manner. The aortic arch, however, was on the right side, as was the descending aorta. Arising from the aortic arch, from before backward, were the left common carotid, the right common carotid and the right subclavian arteries. A ductus arteriosus which measured 1.5 cm. in circumference ran from the right aortic arch at a point beyond the origin of the right subclavian artery to the right pulmonary artery. The left pulmonary artery gave rise to a narrowly patent ductus arteriosus from which the left subclavian artery arose. In view of the arterial relations and connections, there was no undue compression of either the esophagus or the trachea by the anomalous vessels.

The heart had no major malformations. The ventricular septum was intact. In the atrial septum there were two small defects. The valve of the foramen ovale was perforated in several places, each opening being less than 1 mm. in diameter, which allowed communication between the two atria. The atrioventricular valves were normal except for one focus of telangiectasis involving the septal leaflet of the tricuspid valve. The pulmonary valve was bicuspid. There was one leaflet in the left position and the other in the right anterior position. A raphe ran from the pulmonary trunk onto the sinus side of the center of the right anterior leaflet. The aortic valve was normal and both coronary arteries arose from the aorta. The lungs were edematous.

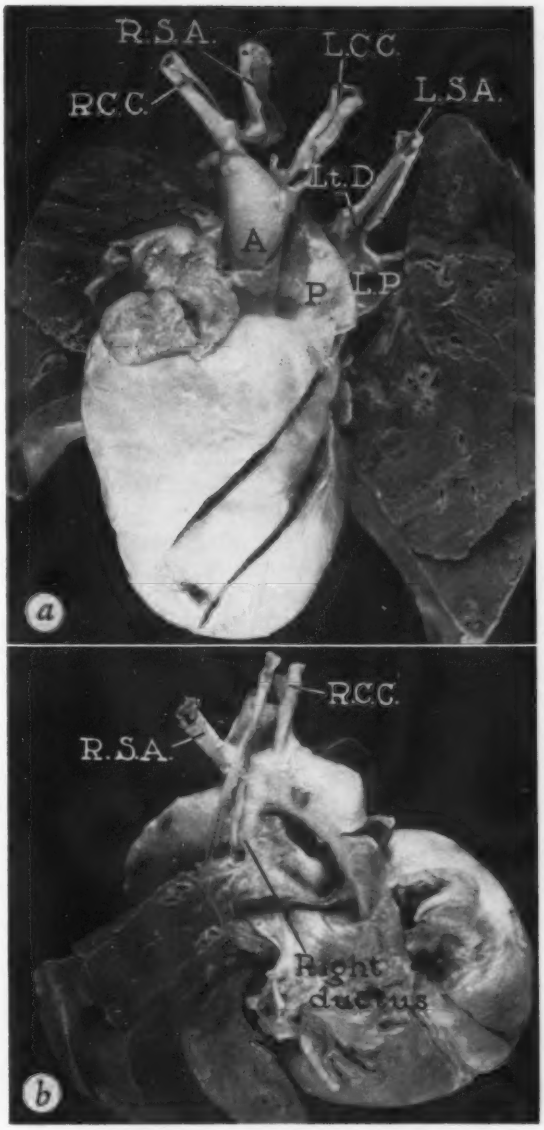

Fig. 2. (*a*) Heart, lungs and great vessels viewed from the front. The ascending aorta (A) and the pulmonary trunk (B) are properly interrelated. The aortic arch is on the patient's right. Arising, from before backward, from the arch are the left common carotid (L.C.C.), the right common carotid (R.C.C.), and the right subclavian (R.S.A.) arteries. The left subclavian artery (L.S.A.) arises from the left pulmonary artery (L.P.) as a continuation of the left patent ductus arteriosus (Lt.D.). (*b*) The heart, great vessels and lungs viewed from the right. Opposite and slightly distal to the origin of the right subclavian artery is the right ductus arteriosus which connects the aorta and the right pulmonary artery. The recurrent laryngeal nerve branching from the vagus nerve is seen to hook around the posterior aspect of the right ductus arteriosus.

COMMENT

The immediate cause of death in this case is not entirely clear. The edema of the lungs may have represented a manifestation of failure of the left ventricle due to a patent ductus arteriosus. Ordinarily, this condition does not give rise to failure so early in life but it may do so in rare instances, as possibly it did in this case. The prominence of the pulmonary hilar vessels may have been a consequence of the widely patent right ductus arteriosus.

The chief purpose of our report is to

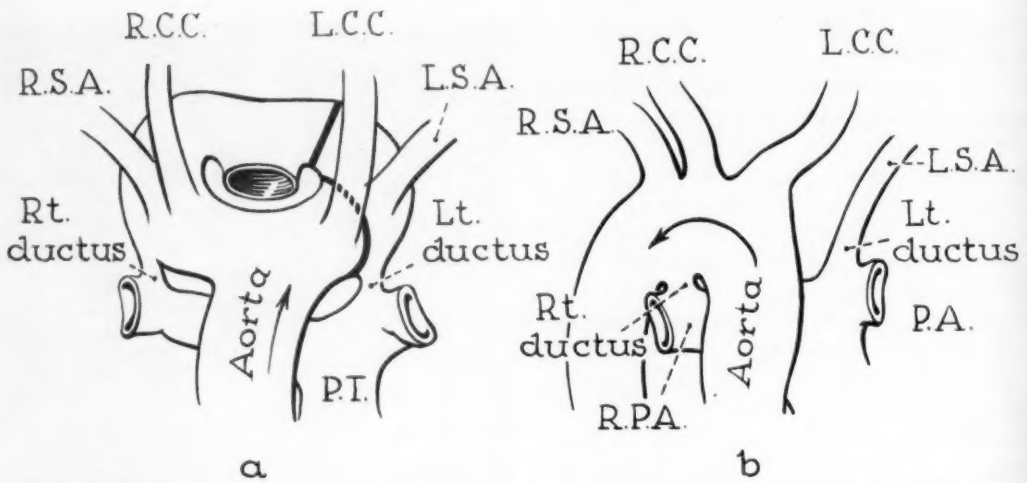

FIG. 3. (*a*) A hypothetical double aortic-arch pattern showing bilateral aortic arches and bilateral ductus arteriosus. The arrangements of the great vessels present in this case, and illustrated in *b*, are explained by interruption of the left aortic arch in two places as follows: (1) in the region between the origins of the left common carotid and the left subclavian arteries and (2) distal to the aortic insertion of the left ductus arteriosus. This leaves the left subclavian artery with a small segment of the left aortic arch only in communication with the pulmonary arteries by way of the patent left ductus arteriosus. (*b*) Arrangements of the great vessels in the case reported.

place on record another example of bilateral ductus arteriosus.

The configuration of the aortic-arch system in this case is like that in the case described by Ghon except that in his case each ductus arteriosus was closed while in our case each was patent, the left being narrowly patent and the right widely patent. In Ghon's case the heart showed a ventricular septal defect and right ventricular infundibular stenosis.

In a previous report dealing with developmental explanation for malformations of the aortic-arch system, Edwards employed a diagram of a hypothetical double aortic arch in which there is an aortic arch and a ductus arteriosus on each side (Fig. 3*a*). The descending aorta is the midline. Each ductus arteriosus runs from the corresponding pulmonary artery to the homolateral aortic arch, the ductus arteriosus inserting distal to the subclavian artery arising from that aortic arch. A common carotid artery for each side arises proximal to the subclavian artery from the respective aortic arch. With this hypothetical scheme (Fig. 3*a*) the malformations present in our case may be explained. By deviation of the descending aorta to the right and by interruption of the left aortic arch at two levels, one between the left common carotid and left subclavian arteries and the other distal to the insertion of the left ductus arteriosus, and allowing for alterations with growth, the left common carotid artery would then arise as the first branch of the right aortic arch. The left subclavian artery would lose its proximal and distal connections with the aorta except for that segment of the left arch that receives the left ductus arteriosus. This would result in the picture seen in our case, in which the left subclavian artery arose as an apparent continuation of the left ductus arteriosus. While there is apparent continuity between the left ductus arteriosus and the left subclavian artery, it is evident from the representation in Figure 3*a* that an element of the left aortic arch lies incorporated in the vascular structure, the proximal part of which is the left ductus arteriosus and the distal part of which is the left subclavian artery.

In our case, in spite of serious derangements in development of the aortic-arch system, there was no apparent abnormal compression of either the esophagus or the trachea. In a strict sense the case cannot be classified as an example of a vascular ring.

Yet from a developmental viewpoint it is related to those malformations that either encircle or compress the trachea and esophagus. This case, in which there was retention of the ductus arteriosus on each side, may be considered a link in the story in which the various configurations that form vascular rings are related to primitive stages in the aortic-arch system.

We should mention the blood flow in the left subclavian artery in our case. Superficially, it might be interpreted that blood would have flowed from the pulmonary arterial system through the left ductus arteriosus into the left subclavian artery. Although no measurements were made to test this point, the above interpretation may be questioned. Since resistance to flow in the left arm may be considered to have been at normal systemic levels, it may be doubted that a sufficient head of pressure was built up in the pulmonary arterial system to force blood through the narrow left ductus arteriosus into this artery.

It is probable that the entire supply for the left subclavian artery came from the right subclavian artery and upper thoracic aorta by way of collaterals. This would be comparable to the situation in coarctation of the aorta wherein one subclavian artery arises proximal and the other distal to the coarctation. Here the subclavian arising from the "low pressure compartment" below the coarctation receives its supply not from the aorta but rather from the contralateral subclavian artery which arises proximal to the coarctation.[9] In such cases blood may flow into the aorta through the subclavian vessel arising below the coarctation.

If such phenomena of flow pertain in our case, it is probable that the left subclavian artery did not receive blood from the pulmonary arteries through the left ductus arteriosus but rather that it supplied blood to the pulmonary arteries through this route.

SUMMARY

We have reported a case of bilateral ductus arteriosus associated with a right aortic arch and right-sided descending aorta and with origin of the left subclavian artery from the left ductus arteriosus. The immediate cause of death was possibly related to the wide patency of the right ductus arteriosus.

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LEFT AORTIC ARCH AND RIGHT DESCENDING AORTA

CASE REPORT

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AS FAR as we have been able to determine, this is the fourth reported case of a left aortic arch and a right descending aorta. Before 1948, this aortic anomaly remained hypothetical. Even though congenital anomalies of the aortic arch were known to occur many years ago,[3,12] it was not until 1950[13] that they were classified into groups, including hypothetical aortic anomalies, depending on the position of the ligamentum arteriosum and descending aorta. Unfortunately this complete classification could not be used by the radiologist because the diagnosis of aortic anomalies is never so complete as to permit determination of the position of the ligamentum arteriosum. This structure has even been missed at the time of surgery.[16,23]

For the radiologist, the following classification will prove useful.[2] Points of reference are the position of the aortic arch and the descending aorta. There are six possible types (Table 1).

Double aortic arch or anomalies of the other aortic branches are not included.

We employ this classification prior to surgical intervention or before knowing the pathologic findings, since fluoroscopy and esophagraphy only show the position of the arch of the aorta, the presence or absence of retroesophageal segments, and at times the position of the descending aorta.

Three general rules may be followed as regards the course of the aorta:

1. Whenever the aortic arch and descending aorta are on different sides of the spine, there is one retroesophageal segment of the aorta (Type 3 and 4).

TABLE I

Aortic Arch	Descending Aorta	Types
left	left	1. normal type 2. normal with retroesophageal right subclavian artery
	right	3. circumflex or posterior
right	left	4. circumflex or posterior
	right	5. right anterior arch 6. right anterior with left retroesophageal subclavian

2. Whenever the aortic arch and descending aorta are on the same side of the spine, no retroesophageal segment is present (Type 1 and 5), but the retroesophageal course of the subclavian vessel of the contralateral side is possible (Type 2 and 6). An exception to this is a case presented by Biederman in which there was a left retroesophageal diverticulum at the junction of the right aortic arch and a right descending aorta, which was the result of an interrupted left aortic arch between the origin of the left common carotid and left subclavian artery.
3. Whenever the descending aorta is rightsided, the lower segment crosses to the left side about the level of the eighth thoracic vertebra.

An interesting case was published recently[16] which could not be well classified since the aortic arch was on the left and the descending aorta was over the midline of

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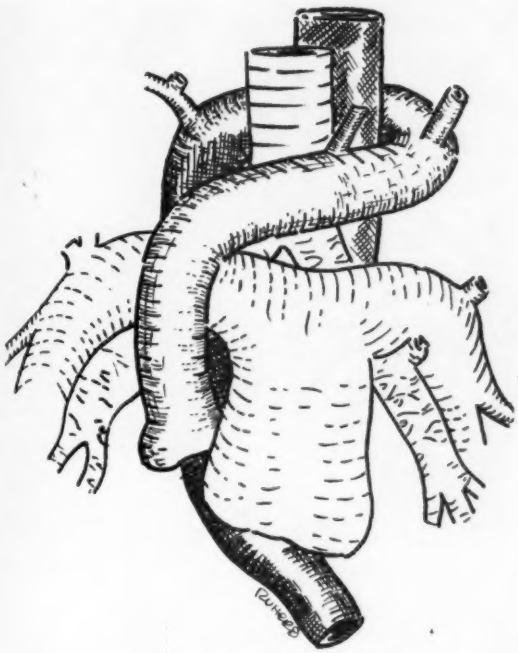

Fig. 1. Schematic drawing after Balsac of the anterior view of the anomaly presented.

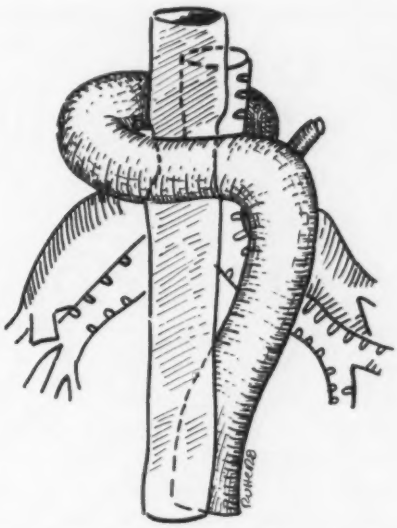

Fig. 2. Schematic drawing of the posterior view.

the spine. There was no retroesophageal segment and the left subclavian artery crossed to the right between the esophagus and trachea. This case probably would represent a Type 2 variation.

Our case falls in subgroup C by using Edwards' classification and corresponds to Type 3 in Table 1. It was considered to be a left aortic arch with a right descending aorta or a left circumflex arch. This anomaly is the result of an interruption of the right aortic arch between the origin of the right subclavian and right carotid artery[7] or posterior to the right subclavian arteries.[16]

The first report of this anomaly was made by Paul who described a seven year old white female and an eleven and one-half year old white male, both of whom were symptomatic, markedly incapacitated, and with evidence of a concomitant cyanogenous congenital anomaly. Edwards' only case was that of a male, seventeen months old, who had symptoms of dysphagia, and an imperforate anus.

Our case is that of a fifty year old white male with no other recognizable anomalies. He is asymptomatic and was diagnosed during routine fluoroscopy of the upper gastrointestinal tract.

The posteroanterior roentgenogram showed the silhouette of the heart and great vessels to be grossly similar to that found in patients of the same age group, with slight arteriosclerotic changes of the aorta manifested by increased density of structure. Careful study in this projection and others revealed that the prominence and contrast of the aortic knob and arch

Fig. 3. Chest roentgenogram, posteroanterior projection.

were due to the overlapping of the anterior and retroesophageal limb of a horizontal left aortic arch. The density of the aorta permitted following of the retroesophageal segment from the left to the right lateral aspect of the sixth thoracic vertebra. This course was horizontal. The aorta then turned downward and descended on the right side for three or four vertebral bodies and crossed back to the left side of the midline, at the level of the ninth and tenth thoracic vertebrae. The esophagus descended in the midline and was not part of the left contour of the heart. The main pulmonary artery and its left branch were at the left of the esophagus. The descending aorta occupied the right half of the anterior aspect of the thoracic spine rather than the right side of the spine.

In the left anterior oblique projection, the ascending aorta turned posteriorly, forming a sharp angulation at the superior portion of the anterior contour of the heart. The normal anteroposterior arch with an inferior concavity was replaced by the straight anteroposterior vessel that suddenly changed course from the left to the right side of the spine. This retroesophageal segment was seen on end in this projection. The distal portion of the arch reached the right side of the spine. The retroesophageal portion displaced the trachea and esophagus anteriorly, and a very slight impression was noted on the posterior wall of the esophagus, but in general, did not change its course markedly.

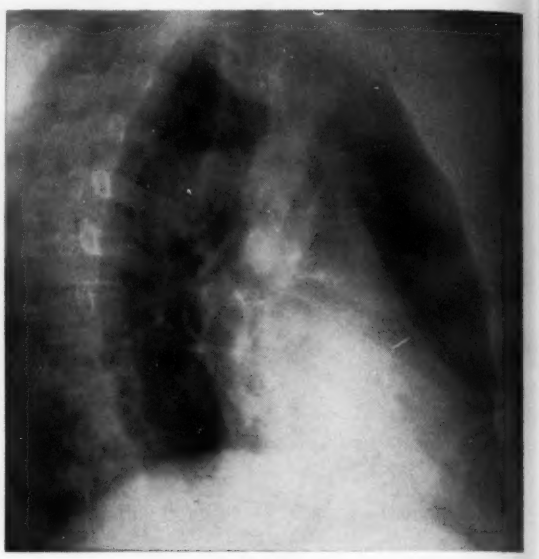

Fig. 5. Right anterior oblique projection.

The right anterior oblique roentgeno-

Fig. 4. Left anterior oblique projection.

Fig. 6. Right lateral view.

Fig. 7. Esophagram, posteroanterior projection.

gram again showed the sharp angulation produced by the abrupt change in direction of the aortic arch in surrounding the esophagus and crossing behind it to descend at the right side of the spine. The trachea and esophagus were displaced anteriorly.

The right lateral projection revealed a wide open square aortic window limited anteriorly by the vertical segment of the ascending aorta. The horizontal and straight anteroposterior segment of the aortic arch constituted the superior margin and the descending aorta, overlying the anterior aspect of the thoracic spine as the posterior border. The retroesophageal segment was not seen in this view.

The impression of the aorta upon the esophagus and the minimal deviation of the trachea and esophagus caused by this vessel in our case was demonstrated on the spot roentgenogram obtained of the esophagus in the upright left anterior oblique position. (Notice the round impression of the retroesophageal aorta on the posterior wall of the doubly contrasted esophagus.)

DISCUSSION

Fluoroscopy and routine roentgenograms of the chest were of diagnostic value in this case of a left aortic arch with a right descending aorta because of the increased density due to arteriosclerotic changes in

Fig. 8. Spot esophagram, left anterior oblique projection, upright position.

the aorta of an adult. This anomaly is included in subgroup C of Edwards' classification, or Type 3 of the given roentgenologic classification.

The impression of the aorta upon the esophagus produced minimal change, consisting only of an indentation on the latter. In contradistinction to the findings of Paul, no portion of the esophagus formed a part of the left contour of the heart. The esophagus remained in a normal position and was normally indented on its left margin by the aortic arch.

CONCLUSION

This is the fourth case reported of a left aortic arch and a right descending aorta and the only case recognized during routine fluoroscopy of the upper gastrointestinal tract in an otherwise asymptomatic adult. Diagnostic features of this anomaly are described and compared with those in cases reported previously.

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ANEURYSMS OF THE SINUS OF VALSALVA

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IT IS the purpose of this communication to review the literature on the subject of aortic sinus aneurysms. The three small dilatations of the aorta located immediately above the aortic valve cuffs, known as the sinuses of Valsalva or aortic sinuses, are almost entirely intracardiac. Aneurysms developing in this location may extend into the cardiac chambers and many encroach upon the pulmonary valve, the atrioventricular valves, the conducting mechanism, the pulmonary or coronary arteries, or they may lead to aortic regurgitation by deforming the aortic ring. In addition, they may rupture into one of the cardiac chambers, the pulmonary artery, or the pericardium.

Aortic sinus aneurysms may be either congenital or acquired. Congenital aneurysms have been described by Taussig,[17] Abbott,[1] and Goehring.[5] These are usually small, finger-like projections or diverticula arising from the sinus, whereas acquired aneurysms may be much larger and often are associated with diffuse dilatation of the sinuses. Congential aneurysms often produce complicated, bizarre physical findings, various authors[12,15,16] having written upon the difficulty of making an accurate diagnosis during life.

Acquired aneurysms are usually syphilitic in type.[13] However, aneurysms in this area may be also arteriosclerotic,[11] or can be due to bacterial endocarditis, or may be atheromatous in nature.

The following patient was examined roentgenologically and certain interesting features noted. Autopsy revealed rupture of an aneurysm of the right sinus of Valsalva into the right ventricle of the heart.

REPORT OF CASE

The patient was a fifty-six year old colored male who was first admitted to the Mississippi Baptist Hospital on February 21, 1951 with a complaint of pain in his chest of one week's duration. The pain was located in the right side of his chest anteriorly and had been preceded by an upper respiratory infection which had partially subsided. It was stated by the patient that he had been rejected by the Army several years previously on the basis of roentgen evidence of a right-sided enlargement of the heart, although he had been asymptomatic at that time and had been accepted for employment as a laborer in shipyards during World War II.

Physical examination disclosed that a large area of dullness existed to the right of the sternum. In this area heart sounds were more clearly audible than at the left apex. Actually, heart sounds were heard over the entire chest with a loud, rumbling systolic murmur being present in early systole, followed by a harsh decrescendo and a split-second sound. The patient had previously had an arrhythmia but had been placed on temporary digitalis therapy with the rhythm being regular at the time of admission to the hospital. Blood pressure was 140/80. Tenderness was elicited over the right nipple area when manipulating the right pectoralis major muscle. Electrocardiographic findings were not helpful. Serologic tests on February 24, 1951 showed a 4 plus Kline reaction with a 2 plus Wassermann reaction being noted.

Fluoroscopy and films (Fig. 1, 2 and 3) of the heart and lungs revealed the presence of a large mass adjacent to the right side of the heart anteriorly which could not be separated from the heart. The left ventricle and aorta pulsated actively, while the large mass itself did not appear to pulsate except for transmitted pulsation. A study of roentgenograms disclosed that calcium was present within the walls of the mass. The possibilities considered were cardiac aneurysm, dermoid cyst, and pericardial cyst with calcification.

Because of the severe pain in his chest and right shoulder which the patient was having and because he was a good surgical risk, the possibility of exploration was considered on the chance that the shadow might prove to be a

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Fig. 1. Aneurysm of the sinus of Valsalva. Note extension into region of right cardiophrenic sinus.

Fig. 2. The anterior location of the aneurysm is shown well in lateral projection.

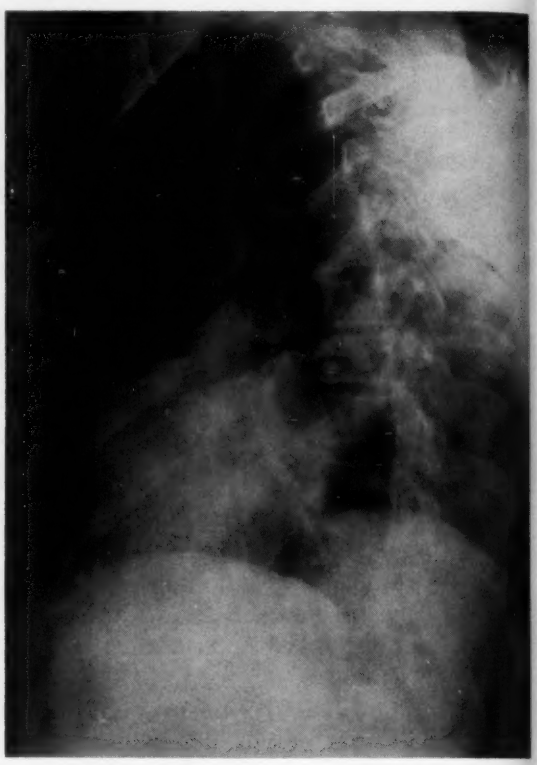

Fig. 3. Note the presence of calcium within the wall of the aneurysm in this left anterior oblique view as well as in other projections.

dermoid cyst, but the patient was discharged from the hospital. On March 28, 1951 he was re-admitted with severe pain in his right chest and severe dyspnea. Progressive fall in blood pressure occurred rapidly with circulatory collapse developing. It was believed upon examination at this time that the patient had ruptured an aneurysm. He died on March 29, 1951, approximately thirty-six hours after admission to the hospital, and less than forty-eight hours after the onset of symptoms.

Necropsy by Dr. F. A. Bratley revealed a concentrically enlarged heart, weighing 1,300 grams, which, together with the aneurysm, was boot shaped. The aneurysm arose from the right sinus of Valsalva with its neck measuring 2 by 3 cm. The mass bulged into the right ventricle along the right lateral wall, the entire aneurysm measuring 8 by 9 by 11 cm. There was a perforation into the right lateral wall of the right ventricle, the defect measuring 9 by 10 mm. and having ragged borders. A section (Fig. 4) through the wall of the aneurysm disclosed apparent destruction of the elastic tissue with the media showing increased vascu-

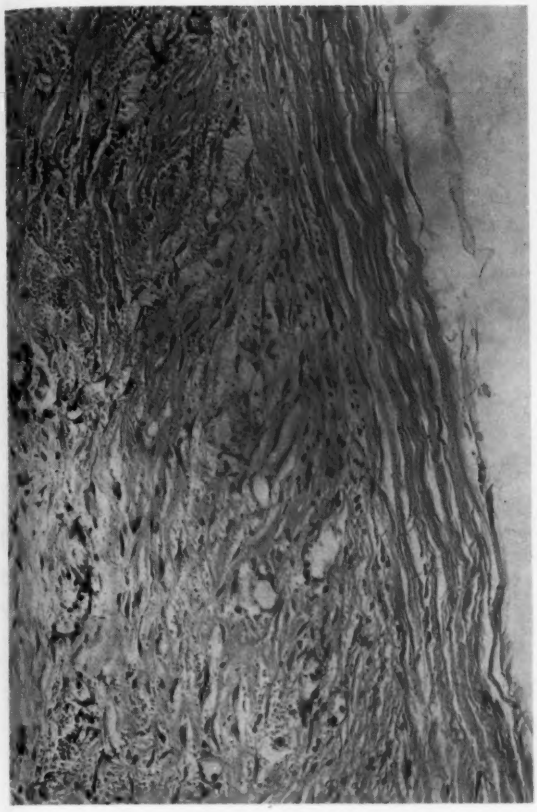

FIG. 4. Photomicrograph of aneurysmal wall.

larity. Occasional small vessels showed perivascular cuffing with lymphocytes.

ANATOMY

The sinuses of Valsalva constitute the space just above the aortic ring, bounded by the aortic leaflets and the inner surface of the heart. This portion of the aorta is called the sinus aorta while the aorta immediately above is called the supra-sinus aorta. Various systems of nomenclature are used but the sinuses may be named as follows: First, the left lateral sinus above which is found the orifice of the left coronary artery and thus may also be called the left coronary sinus; second, the anterior or septal aortic sinus above which is found the orifice of the right coronary artery and thus may be called the right coronary sinus. This sinus occupies the upper border of the posterior aspect of the left ventricular surface of the interventricular septum; third, the right lateral sinus which has no coronary orifice above it and which, therefore, may be called the non-coronary sinus.

In their excellent monograph, Jones and Langley[9] state that Warmsley in 1929 had pointed out that the standard nomenclature was based on the assumption that one aortic sinus lay anteriorly and two posteriorly with the right coronary sinus lying anteriorly. However, in 15 per cent of the cases, the right and left coronary sinuses may lie anteriorly while the non-coronary sinus lies posteriroly. For this reason it might be preferable to name the sinuses by their relation to the coronaries.

The coronary sinuses are related to the pericardium and great transverse sinus superiorly and also have intracardiac relations. The anterior or right coronary sinus directly underlies the right surface of the interventricular septum. The right lateral sinus, or non-coronary sinus, is in the proximity of the upper anterior portions of the right and left atrium. The anterior half of the sinus is adjacent to the cavity of the right auricle, while the posterior half of the sinus is related to the cavity of the left auricle. The anterior half of the left lateral or left coronary sinus is adjacent to the pulmonary septal sinus and pulmonary orifice. The posterior half of the left lateral sinus has extracardiac relations, being situated between the base of the left ventricle near the base of the left auricular appendage and the beginning of the left anterior interventricular artery.

PATHOLOGY

The difficulty in deciding whether a given aneurysm shown at autopsy is congenital or acquired has been pointed out. This is particularly apparent where there is associated bacterial endocarditis. Congenital aneurysms are believed to arise from defective development of the distal bulbar septum. Congenital weaknesses here may develop at points of fusion. All of the aortic sinus aneurysms collected by Abbott[1] in 1919 arose from the right coronary sinus with aneurysms from the non-coronary sinus being subsequently reported. There is no reported instance of a con-

genital aneurysm arising from the left coronary sinus. These aneurysms can often be diffentiated from the acquired type by the age and history of the patient. The most important etiologic factor in the acquired type of aneurysm is syphilis of the aorta.

When hypoplastic syphilis involves the aorta, the resultant thinning of the media forms the groundwork for the saccular aneurysm. The lesion is usually discrete so that one sinus or merely a part of it may be involved. Luetic aneurysms attain a much larger size than congenital aneurysms, the congenital aneurysms usually not causing a bulging of the external surface of the heart. At times the sinuses may be involved by an extension downward from the supra-sinus aorta. Harris and Schattenberg[6] reported a case of rupture of a supra-sinus aneurysm into the right ventricle with small aneurysms being present also in each of the sinuses, these being on a leutic basis.

CLINICAL DISCUSSION

From a study of the above described anatomic relations, it is apparent that there are numerous possibilities for unusual findings and that no particular physical sign or symptom can be designated as diagnostic of these lesions. Jones and Langley[9] have collected 25 reported cases of congenital sinus aneurysms proved at necropsy, together with 22 acquired aneurysms confined to the aortic sinuses and confirmed pathologically. The congenital aneurysms reported were confined to the right coronary sinus and adjacent two-thirds of the non-coronary sinus. The aneurysms were always small but, owing to their thin walls, commonly rupture to form cardio-aortic fistulas, usually with the communication existing between the right coronary sinus and the right ventricle, or between the non-coronary sinus and the right auricle. Although the aneurysms remain intracardiac and do not affect extracardiac structures, or rupture outside the heart, they frequently do cause disturbances of intracardiac structures, especially the pulmonary and tricuspid valves. Congenital sinus aneurysms are nearly always associated with other developmental anomalies, usually anomalies of the aortic valves or ventricular septal defects.

When rupture occurs, an abrupt onset of signs and symptoms is a characteristic part of the clinical syndrome. Those which are congenital in origin usually rupture in early adult life, between fourteen and thirty years of age, while those due to syphilis occur at a somewhat later age. Pain may or may not be an outstanding feature at the time of rupture, depending upon whether coronary occlusion, due to involvement by the aneurysm at the coronary orifice, is associated.[4] The history, particularly in the congenital cases, is usually that of a patient who has previously been in excellent health and suddenly develops signs and symptoms of cardiac difficulty. Dyspnea is likely to develop abruptly after the rupture of the aneurysm and becomes progressively more severe. The pulse pressure becomes wide. The heart is usually enlarged regardless of etiology. Insofar as murmurs and thrills are concerned, all observers agree that the murmur and thrill are continuous through systole and diastole, and are usually quite loud. Rapid progression of signs and symptoms is the rule. Although one patient lived for nine years with a congenital aneurysm, the condition is usually fatal within two or three months after the time of rupture.

The clinical syndrome varies considerably, depending upon the site of rupture. Herrmann and Schofield[7] have reported the syndrome of rupture of aortic sinus aneurysms into the right atrium. Rupture of these aneurysms into the right atrium has also been reported by Higgins[8] in 1934, by Wright[21] in 1937, and by Kawasaki.[10,11] Venning[19] has described 7 cases of sinus of Valsalva aneurysms, in one of which he made a correct antemortem diagnosis of rupture into the right atrium.

In addition to the report of rupture of an aneurysm of the aorta into the right ventricle by Harris and Schattenberg,[10] rup-

ture into the right ventricle has also been reported by Schwab and Sanders[14] and by Tompkins.[18] Venning states that the correct diagnosis of rupture into the right ventricle has been made at least seven times: By Scott, White, Porter (twice), by Herrmann and Schofield (twice), and by Tompkins. Scott[15] reported a case of an aortic aneurysm rupturing into the pulmonary artery. Boyd[3] states that rupture most commonly takes place into the pericardium, less commonly into the superior vena cava or pulmonary artery, and rarely into the chambers of the heart.

ROENTGENOLOGIC CONSIDERATIONS

In studying cases in which aneurysms are present roentgenologically, one must bear in mind that, because of the anatomic relations, all aortic sinus aneurysms are located within or associated with the cardiac silhouette. Consequently, when one encounters an intracardiac bulging or a mass which cannot be separated from the heart, the possibility of an aneurysm of the sinus of Valsalva should be suspected.

Aneurysms of the anterior or septal aortic sinus would usually bulge through the interventricular septum and extend anteriorly into the conus of the right ventricle. These aneurysms usually present forward and a bulge in the cardiac silhouette would be most noticeable in anterior projection, but only after considerable enlargement has taken place. Aneurysms of the posterior half of the right lateral sinus would likely extend into the left atrium, producing a bulge in the retrocardiac space, best seen in the right oblique position or showing pressure on the barium-filled esophagus. Aneurysms of the anterior half of the right lateral sinus of Valsalva would also bulge into the right atrium and perhaps extend into the right cardiophrenic sinus. With aneurysms of the anterior half of the left lateral sinus, encroachment upon the pulmonary septum is present and a bulge may be seen below the pulmonary area on the left. On the other hand, aneurysms of the posterior half of the left lateral sinus may present a bulge in the supracardiac space on the left, it being recalled that this sinus has extracardiac relations.

It is obvious that these bulges of greater or lesser degree will be more likely detected by careful fluoroscopy while rotating the patient. In addition, when rupture has occurred but has not been fatal, fluoroscopy may aid in showing expansile pulsations of the small branches of the pulmonary arteries, similar to that seen in cases of atrial septal defect. It was by this means that Venning made his correct antemortem diagnosis of rupture of a right sinus of Valsalva aneurysm into the right auricle.

In the case reported in this communication, several significant features were noted: First, it was observed on the roentgenograms that calcium was present within the lining of the aneurysmal sac. In reviewing the cases in the literature no description of the presence of calcification within these aneurysms was found. Actually, calcification in the wall of the cardiac aneurysms, as mentioned by Bogoch and Christopherson[2] has been considered a relatively uncommon occurrence. Because of the presence of the calcium, it was difficult to differentiate between aneurysm and cyst, either of the dermoid type or of the pericardial type. Second, no pulsation, except transmitted, was elicited. The absence of pulsation is obviously the result of organization. Third, because of the location within the right cardiophrenic sinus, the possibility of an abdominal herniation should also be considered in the differential diagnosis. Fourth, the race and occupation of this patient should be noted, it having been pointed out by White[20] that aneurysms are found six to eight times more often in colored than in white people, not only because of the greater incidence of syphilis in Negroes but probably also because of its less satisfactory treatment among them and the heavy type of labor engaged in, it being considered that stress and strain contribute to the formation of aneurysm.

SUMMARY

The literature concerning aneurysms of the sinus of Valsalva has been reviewed. Rupture of syphilitic aneurysms of the aortic sinus into the right ventricle is a rather rare occurrence but has been described several times in the past. A case showing this complication is reported. The interesting features are that the aneurysm was large, was located in the region of the right cardiophrenic sinus anteriorly, did not actively pulsate, and it contained calcium within its walls. As is usually true when such complication develops, this patient died in less than forty-eight hours following rupture of the aneurysm into the right ventricle.

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The author wishes to express his gratitude to Drs. Hans Karl Stauss and Edward North for use of their clinical records and to Dr. Kenneth Heard for aid in the histopathologic study.

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Vol. 76, No. 4

THE VALUE OF LAMINAGRAPHY IN DIAGNOSIS AND EVALUATION OF COARCTATION OF THE AORTA*

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THE diagnosis of the adult type of coarctation of the aorta is generally considered to be quite exact at the present time. The clinical evaluation of the patient yields a number of physical signs which may suggest the diagnosis. A number of roentgen signs have been enumerated in the past two decades; these make the roentgen diagnosis of the adult type of coarctation relatively simple in the majority of cases.

Rib notching is usually obvious when present. In one series, Pugh[11] found no notching in 12 of 18 patients. He felt that on this basis the diagnosis may not be left to the radiologist. Although Roesler[12] states that rib erosion is the most unequivocal sign of coarctation, several papers have appeared in the literature describing cases with rib notching in which the notching was due to other processes. Gladnikoff,[6] citing Laubry and Heim de Balsac, states that notching may occur in disease of the aortic and mitral valves and in essential hypertension. Batchelder and Williams[2] present a case report of a patient with notching of the left ribs only without coarctation. At autopsy this patient had tetraology of Fallot and the authors concluded that the notching was due to a congenital anomaly of the intercostal arteries.

Levin and Rigler[9] have presented an excellent survey of the literature related to rib notching in the absence of coarctation. They described 8 cases of rib notching following unsuccessful Blalock-Taussig operations and cited obstruction of the third portion of the subclavian artery as the cause of rib notching in their series.

McCord and Bavendam[10] reported 2 cases, one of which had had a large fibrocalcific mediastinal mass and the notching was produced by enlarged, dilated, tortuous anastomosing intercostal arteries. The second case was a luetic with dilated intercostal arteries without coarctation. Green[7] mentions von Recklinghausen's disease as a cause of rib notching in the absence of coarctation.

Among the other diagnostic criteria for coarctation, Figley[5] has listed: (a) inconspicuous ascending aorta; (b) dilatation of ascending aorta; (c) hypoplastic aortic arch; (d) visualization of the site of coarctation in the left anterior oblique position at fluoroscopy or on roentgenograms as a defect in the aortic posterior wall; (e) prominence of the left border of the superior mediastinum on posteroanterior roentgenograms and prominent pulsation of this region when seen fluoroscopically. This is usually attributed to the dilated left subclavian artery; (f) displacement of the esophagus; (g) post-stenotic aneurysm; (h) indentation to the right in lateral margin of the aorta just above the level of the left main pulmonary artery.

The fact that the defect was infrequently seen on the plain roentgenogram made in the left anterior oblique position has placed added emphasis on the laminagraphic study of the aorta. Scott and Bottom[13] found that the method is of definite advantage since it avoids an intravenous injection, it is relatively simple, there is no hazard or discomfort from its use, and the overlying shadows are eliminated. They stress the fact that the best laminagraphic studies of the aorta are those in the older age groups. From the point of view of technique these authors suggest the left anterior oblique position be used for the ascending

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aorta, and the left lateral position to visualize the descending aorta. No patient with coarctation is included among their cases.

Weinbren[15] has not been successful in demonstrating the aortic arch by means of laminagraphy in the left anterior oblique position. He has found that the combination of angiography and laminagraphy, *i.e.*, angio-cardio-laminagraphy gives the best results. Busch[3] advocates the use of laminagraphy in preference to angiocardiography because the contrast medium is diluted and lost in the pulmonary circulation. She feels that while retrograde aortography gives good results, both methods using a contrast medium are time consuming, have a potential mortality of one per cent, and require a specialized team. She describes the findings in 3 patients examined by laminagraphy, in whom the lesion was demonstrated. One illustration of a laminagram made in the left anterior oblique position showing the lesion is presented in her paper.

Even and co-workers[4] used laminagraphy to better define the roentgenographic findings in 6 cases of aortic disease. The lateral and oblique positions proved more valuable than the posteroanterior position. Laminagraphy was used by these authors in preference to kymography, transparietal puncture and intravenous angiography to distinguish between aneurysms and neoplasms.

Shanks[14] points out that laminagraphy sometimes reveals the stenosed area clearly, although he prefers angiocardiography as the ideal method.

In an effort to study the value of laminagraphy in right posterior oblique and left anterior oblique positions as compared to conventional roentgenography and laminagraphy in the lateral position, 16 cases of adult coarctation of the aorta have been reviewed. In 11 of these 16 patients the diagnosis was made clinically. The roentgen findings are listed in Table 1.

TECHNIQUE

In performing laminagraphic examination, speed of tube traverse, distance of tube traverse during exposure of the film, time of exposure, milliamperage, kilovoltage and tube-target distance are important and variable factors. Typically, the patient is placed in 45° left anterior oblique or right posterior oblique position and a plane transecting the manubrium is selected. The radiologist chooses the technique, positions the patient and selects the plane of section. The factors used are: 14 inches tube traverse, 2.5 speed (the most rapid on our attachment), 2.5 second exposure, 68 kv., 30 ma. and 40 inch target-film distance. If the patient is quite large (36-40 cm.) the

Table I

ROENTGEN FINDINGS IN COARCTATION OF THE AORTA. PLAIN ROENTGENOGRAPHY (16 CASES)

	None	1 plus	3 plus	4 plus
Rib notching	6	7	3	
Enlargement of left ventricle	12—moderate to marked			
Prominence of left superior mediastinum	3			
Defect in left anterior oblique position	5			
Small knob of aorta	12			
Esophageal displacement	4 slight			
Laminagraphy (9 cases)				
Lateral Position				
Technically unsatisfactory	3			
Defect not seen	1			
Defect seen	1			
Right Posterior Oblique or Left Anterior Oblique Position				
Defect seen	4			

same relative tube traverse, distance and speed are used, but the milliamperes are raised to 100, and an exposure time of 1.5 seconds is substituted for the previous setting. No more than 4 sections are required. The need for the radiologist to personally perform or supervise the procedure cannot be emphasized too strongly.

REPORT OF CASES

Case I. J. W. T. This was a twenty-five year old white male who had been asymptomatic his entire life except on moderate to marked exertion. In 1943, following induction into the Navy he was found to have a blood pressure of 225/110 in the upper extremities. Blood pressure was unobtainable in the legs. In January, 1953, a clinical diagnosis of coarctation of the aorta was made and he was then transferred to Atlanta Veterans Administration Hospital for angiography and surgery.

Physical examination corroborated the above diagnosis. Roentgenograms showed minimal to moderate rib notching, a small aortic knob, and moderate cardiac enlargement (Fig. 1). A defect in the aorta was thought to be present on the left anterior oblique roentgenogram (Fig. 2). An angiogram was not done. Laminagraphic study of the aorta in right posterior oblique position showed a clear-cut indentation into the posterior surface of the aorta (Fig. 3).

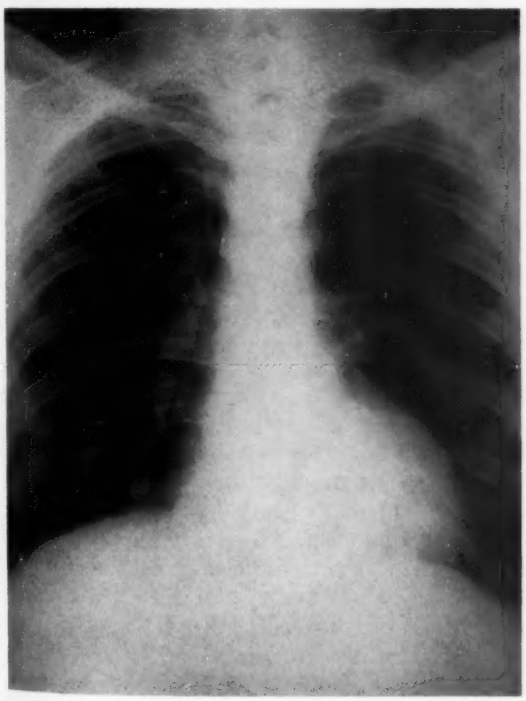

Fig. 1. Case I. Routine chest roentgenogram shows minimal to moderate rib notching, a small aortic knob, and moderate cardiac enlargement.

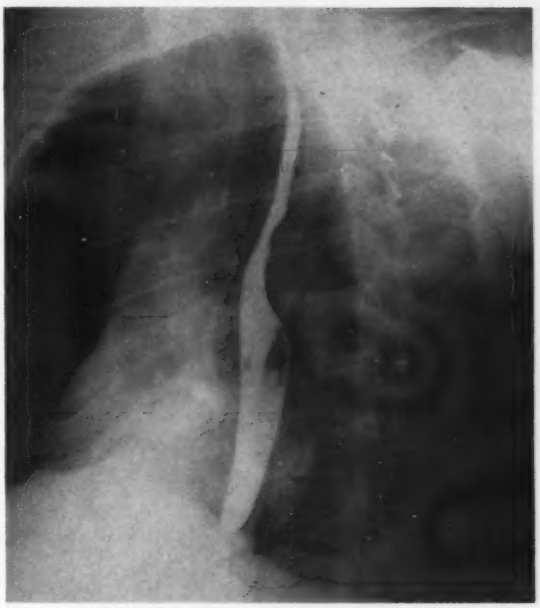

Fig. 2. Case I. Left anterior oblique roentgenogram shows a poorly defined defect in posterior margin of aorta just proximally to left pulmonary artery shadow.

At surgery, the coarcted segment was resected and a primary anastomosis of the aorta done. The procedure was well tolerated and the patient left the operating room in good condition. Good femoral and pedal pulses were noted immediately.

Case II. This was a twenty-seven year old white male who was referred to the Atlanta Veterans Administration Hospital in January, 1953, because of an "anomaly of the aorta" discovered when he developed the "flu" six days previously. He was not accepted for military service in 1943, because of high blood pressure, but in 1944 he was drafted into the service. At the age of ten he noted pains in the legs at night. Recently, while working as a grocery clerk, he noted left chest pains every two to three days. There was mild dyspnea on exertion.

Physical examination disclosed prominent carotid pulsations and firm, tortuous radial arteries with prominent pulsations. There were no pulsations palpable in the abdominal aorta or in the vessels of the lower extremities. The

Fig. 3. Case I. Right posterior oblique laminagram clearly shows clear-cut indentation into the posterior surface of the aorta.

sclerae were slightly icteric. Laboratory data indicated a serum bilirubin of 5.8 mg. per cent, a cephalin flocculation of 4 plus in 72 hours, and a thymol turbidity of 12.3 units. A clinical diagnosis of infectious hepatitis and coarctation of the aorta was made.

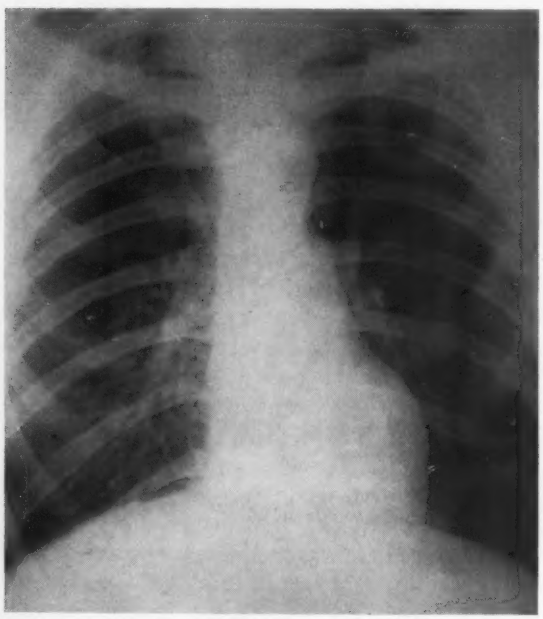

Fig. 4. Case II. Routine chest roentgenogram shows rib notching, small aortic knob and slight left ventricular enlargement.

Fig. 5. Case II. Left anterior oblique roentgenogram shows suggestive defect in aorta.

Roentgen studies showed rib notching, a small aortic knob, and slight left ventricular enlargement (Fig. 4). A suggestive defect in the left anterior oblique roentgenogram was noted (Fig. 5). Laminagraphy in right posterior oblique position clearly showed a smooth depression in the posterior surface of the aorta just above the left pulmonary artery (Fig. 6). Angiograms were not made. The patient was discharged on March 18, 1953, to be recalled in six months. He failed to return for operative correction of the aortic anomaly.

Case III. W. M. This was a thirty-two year old white male who in 1943, while in military service, noted irregular heart action. This irregularity lasted several minutes and was accompanied by mild dyspnea. Hypertension was discovered in 1943, and a diagnosis of coarctation of the aorta made after roentgenographic and electrocardiographic studies were completed. He was then discharged from the military service. Shortly thereafter he began to have mild exertional dyspnea, slight pedal edema and occasional orthopnea. Physical examination on admission showed weak femoral

Fig. 6. Case II. Right posterior oblique laminagram shows smooth depression in posterior surface of the aorta just proximally to the left pulmonary artery.

pulsations and absent pulsations below this area.

Roentgenograms disclosed a shell-like zone of calcification in the projection of the distal aspect of the aortic arch. Moderate rib notching was seen (Fig. 7). A smooth depression was noted in the left anterior oblique position on the posterior aspect of the left side of the aorta just above the calcified density (Fig. 8). Laminagraphic study corroborated this finding and suggested aneurysmal dilatation of the aorta below the coarcted segment (Fig. 9). An angiocardiogram and retrograde aortogram confirmed the aneurysmal widening of the aorta below the coarcted segment (Fig. 10). The patient refused operation.

Case IV. G. C. This was a twenty-four year old mechanic who was well until February, 1950, when he noted the onset of sharp precordial pain with pleuritic radiation, dyspnea, and aching in joints and muscles. After several hospitalizations he was sent to the medical center at Fort Sam Houston, Texas, with a diagnosis of acute rheumatic fever. Blood cultures

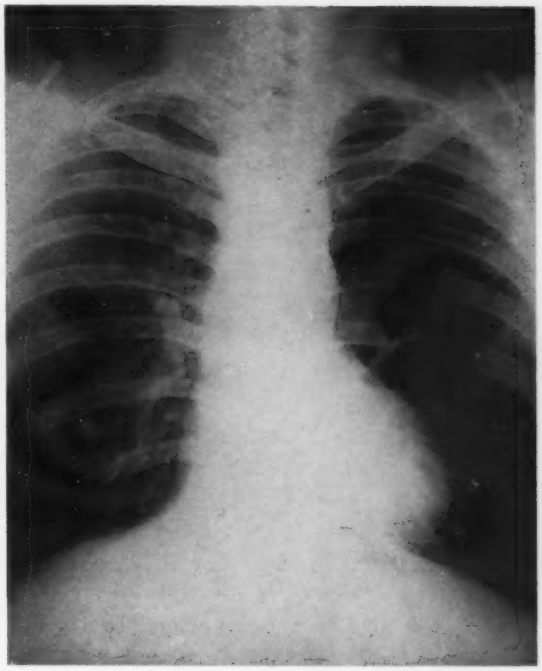

Fig. 7. Case III. Routine chest roentgenogram shows rib notching and a shell-like plaque of calcium in the projection of the distal aspect of the aortic arch.

Fig. 8. Case III. Left anterior oblique roentgenogram discloses a smooth depression in the posterior aspect of the aorta just above the shell-like area of calcification.

Fig. 9. Case III. Left anterior oblique laminagram corroborates the findings of Figure 8, and discloses suggestive aneurysmal dilatation of the aorta below the coarcted segment.

were positive for alpha streptococcus, and he was treated for five weeks with 2,400,000 units of penicillin. During this hospitalization examination disclosed no pulsations in the legs. He was discharged from the service as having congenital heart disease with a bicuspid aortic valve. He was subsequently seen by an intern-

Fig. 10. Case III. Retrograde aortogram reveals site of coarctation and the aneurysm below this point.

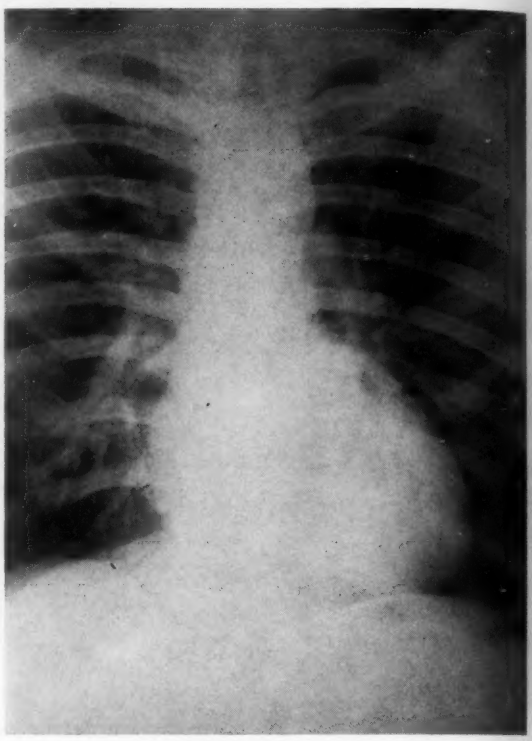

Fig. 11. Case IV. Routine chest roentgenogram shows moderate left ventricular enlargement and rib notching. An ovoid shadow projects through the concavity of the aortic knob and left heart border.

ist who made the diagnosis of coarctation of the aorta.

Physical examination disclosed marked cardiac enlargement and a loud systolic murmur best heard over the aortic area radiating toward the apex. No palpable pulsations were present in the lower extremities.

Roentgen studies demonstrated moderate left ventricular enlargement. An ovoid shadow 3 by 4 cm. in dimension projected through the concavity between the aortic knob and the left heart border in the posteroanterior view (Fig. 11). The possibility that this represented an enlarged pulmonary artery was considered.

Laminagraphy in left lateral position revealed a sharply defined bulge in the aorta just below a clear-cut indentation into the aortic wall 1.5 cm. below the origin of the left subclavian artery (Fig. 12). An indirect aortogram was performed corroborating these findings (Fig. 13).

At operation the site of coarctation was identified and below this an aneurysmal dilatation of the aorta was found; this extended distally

Fig. 12. Case IV. Laminagram shows sharply defined bulge in the aorta just below a definite indentation in its posterior wall 1.5 cm. below the origin of the left subclavian artery.

Fig. 13. Case IV. Retrograde aortogram corroborates laminagraphic findings.

for a distance of 4–5 cm. Resection was deferred in the absence of an aortic graft. An aortic graft was obtained two weeks later but a stitch abscess prevented a second operative procedure. No subsequent operation has been performed.

Case V. W. T. This patient stated that his family was told at his birth that he had a heart defect. He has never had any symptoms and has led an active and normal life. Later in life he had noticed that after exertion his heart would beat quite rapidly. In July, 1948, he had a sudden onset of numbness in all extremities associated with pain around his heart. He had had several of these attacks in sixteen months prior to admission. Physical examination showed the blood pressure in the upper extremities to be 180/110; in the lower extremities 130/100.

The heart was enlarged to 10 cm. laterally from the midsternal line in the sixth left intercostal space. A Grade II systolic murmur was heard over the precordium, maximally over the aortic area. The lower extremity pulsations were quite weak. Laminagraphic study, in right posterior oblique position, through the plane of the aorta demonstrated calcification in the ascending aorta and arch down to a point in the distal part of the arch where the calcification ended (Fig. 14). The ascending aorta was broad, but distal to the origin of the left subcalavian artery it narrowed to one-half

Fig. 14. Case V. Right posterior oblique laminagram shows calcium plaques in wall of ascending and transverse aorta, and extending to the point of coarctation.

its size. Linear calcifications extending off the superior side of the arch appeared to be in the left subclavian or a combination of the left subclavian and the left common carotid. The actual site of the coarctation was thought to be at the point where the calcification ended.

At surgery these findings were corroborated. Left dorsal sympathectomy was done.

DISCUSSION

An analysis of this group of cases discloses that the clinical diagnosis was made in each of the 5 patients prior to the laminagraphic study. Each presented rib notching of a moderate degree. In 2 a defect was suggested in the contour of the aorta on the plain left anterior oblique roentgenogram.

Among the remaining 11 cases of the 16 in this study, rib notching was absent in 6. In an effort to determine the possible or potential value of laminagraphy in right posterior oblique or left anterior oblique position, a review of the clinical and roentgen findings in these 6 cases was made. In 4, a clinical diagnosis of coarctation was established. In 1 patient, the coarctation was discovered at autopsy, the patient having been treated for multiple myeloma. In 1 instance, the clinical diagnosis was hypertension and dissecting aneurysm of the aorta.

Study of the plain posteroanterior roentgenograms in these 6 patients showed that the aortic knob was small in 1 patient and hypoplastic or very small in 3 others. Two of the 6 patients presented no cardiac enlargement. As to a defect in the plain left anterior oblique roentgenogram, in 3 instances no defect was seen. In 1 case the defect was suggested but not clear. Two of the patients showed a clear-cut defect in the posterior wall of the aortic arch. One of the 6 patients presented an accentuated vascular shadow in the medial aspect of the left superior mediastinum, but in the remaining 5 this shadow was not seen. Angiocardiography was not done in 3 and the defect was seen in only 1 of the 3 patients in whom it was performed.

It is probable that if laminagraphy of the aortic arch in left anterior oblique or right posterior oblique position had been carried out in these 6 cases, the diagnosis of coarctation of the aorta would have been corroborated or made on the basis of this examination. It is felt, therefore, that laminagraphic study is of definite value in establishing a diagnosis of coarctation of the aorta. In particular, laminagraphy aids in delineating calcific deposits within the wall of the aorta and its branches at or near the site of coarctation. Leigh[8] and Abbott[1] have found the method useful in the preoperative evaluation of the patient for resectional surgery. The extent of the calcification, its position in relation to the coarcted segment, and the presence or absence of calcification in an aneurysm of the post-stenotic segment or aortic branches are important factors in determining criteria of operability. The nature, relationship, and size of the left subclavian artery can also be ascertained in some cases and an impression as to its size can be gained in others. This knowledge is of interest to the surgeon should he consider a by-pass procedure rather than resectional surgery and aortic graft replacement.

SUMMARY

Laminagraphy in left anterior oblique or right posterior oblique position is of significant value in the roentgen evaluation of the aorta in coarctation.

1. Laminagraphy clearly shows the site of narrowing or constriction of the aorta.
2. The presence or absence of calcification at or near the coarcted segment may be demonstrated.
3. The size and status of the left subclavian artery may be determined in some instances.
4. The method is simple and is devoid of potential mortality to the patient when compared with contrast procedures.

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The author expresses his appreciation to Dr. Ted F. Leigh for making Case v available for this study.

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COARCTATION OF THE ABDOMINAL AORTA*

REPORT OF A CASE ILLUSTRATING THE VALUE OF AORTOGRAPHY AS A DIAGNOSTIC AID

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THE symptoms and clinical findings associated with coarctation of the thoracic aorta are well established. Coarctation occurring at the usual site in the thoracic aorta distal to the origin of the left subclavian artery is easily recognized. Diagnostic clinical observations include hypertension in the upper extremities, diminution or absence of pulsations in the lower extremities, a collateral circulation and deformities of the aorta on routine roentgen studies of the chest. Stenosis in the lower thoracic or abdominal aorta presents added difficulty in diagnosis and localization of the site of the lesion. The purpose of this presentation is to discuss the diagnosis of coarctation of the abdominal aorta by clinical and roentgenologic methods and to emphasize the value of aortography.

REVIEW OF THE LITERATURE

Ten cases of coarctation of the abdominal aorta have been reported to date. The first report of a case was published by Power[8] in 1861. Maycock,[7] in 1937 reported his observations over a five year period in an eighteen year old patient with coarctation of the aorta located 1.5 cm. below the origin of the renal arteries. The patient complained of palpitation, dyspnea and precordial pain on exertion. Physical examination revealed the blood pressure in the arms to be 200 systolic, 89 diastolic, while that of the legs was 130 systolic, 110 diastolic and occasionally was unobtainable. The patient had persistent tachycardia (120/min.), palpable pulses in the internal mammary arteries, and a systolic murmur at the right border of the sternum, heard best at the level of the xiphoid process of the sternum anteriorly. A harsh systolic murmur was also heard posteriorly from the level of the fifth to the tenth dorsal vertebra and along the twelfth rib. There was an extensive collateral circulation involving the seventh, eighth and ninth intercostal, the internal mammary, the epigastric, and the subscapular arteries. Death occurred at the age of twenty-three following a severe amebic infestation.

Autopsy revealed an abrupt diminution in the size of the aorta 1.5 cm. below the origin of the renal arteries, with the formation of a firm cord 3 cm. in length and 8 mm. in diameter. The lumen was completely occluded by a firm gray laminated mass fused to the vessel wall, but the aorta became patent again 4.5 cm. above the bifurcation. Degenerative and arteriosclerotic changes were present in the intima of the aorta proximal to the stenosis, but were absent distally. Maycock regarded this as a case of congenital stenosis of the lower part of the abdominal aorta, with subsequent thrombosis and recanalization.

Baylin[3] in 1939, described a thirty-two year old colored man with obstruction of the abdominal aorta. There was no history to suggest vascular disturbances, but an extensive collateral circulation was present. The obstruction was distal to the orifices of the renal arteries and extended to the origin of the inferior mesenteric artery. This segment of the aorta was irregular, nodular and stony hard, and was obstructed by a calcification which appeared to have developed in aneurysmal dilatations of the aorta. The etiology could not be determined from gross inspection of the lesion.

Steele[9] reported the case of a forty-six year old white woman who complained of

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headaches and dizziness. The blood pressure in the arms was found to be 210–260 systolic, 120–180 diastolic, and that in the legs was 100–120, 90–100. The femoral pulses were barely palpable. Evidences of collateral circulation were present in the lower chest posteriorly. The chest roentgenograms demonstrated characteristic rib notching. The patient died of a cerebrovascular accident. The postmortem examination revealed marked constriction of the aorta at and above the renal arteries.

Bahnson, Cooley and Sloan's[2] patient was a thirty-five year old white woman who complained of headaches, dizziness, exertional dyspnea, and numbness and stiffness of the legs. The blood pressure in the arms was 230 systolic, 110 diastolic, and femoral pulses were palpable. A systolic murmur was heard over the back on both sides of the vertebral column and was loudest at the level of the tenth dorsal vertebra. It was also heard anteriorly at the level of the xiphoid process. There was no evidence of a collateral circulation, and no rib notching was seen on the roentgenograms. The angiogram, however, revealed complete obstruction of the abdominal aorta at the level of the first lumbar vertebral body, just below the origin of the renal arteries. Surgical exploration revealed enlargement of the aorta proximal to the obstruction with diminished pulsations distally.

Kondo and his co-workers[6] described coarctation of the abdominal aorta in a twelve year old Japanese girl with an associated anomaly of the vessels arising from the aortic arch. She complained of lassitude and weakness of three years' duration and edema of the lower extremities. The systolic blood pressure in the left arm was 40. No pulsations were palpable in the right radial artery and both femoral arteries. The seventh to twelfth intercostal arteries were palpable bilaterally and a systolic murmur was heard along the vertebral column posteriorly, being loudest in the paraumbilical region. The chest roentgenogram revealed an enlarged left ventricle and passive hyperemia of the lungs, but no evidence of rib notching. Autopsy revealed a marked coarctation of the abdominal aorta with a valve-like structure just above the renal arteries. The proximal aorta was greatly increased in thickness and contained numerous bright yellow atheromatous deposits. The distal segment was narrowed to about one-fourth the diameter of the thoracic aorta.

Goldzieher and co-workers[5] reported the case of a forty-five year old female with symptoms of headaches, nausea, vomiting and episodes of temporary unconsciousness. The blood pressure in the right arm was 160 systolic, 100 diastolic. There was no record of the blood pressure readings in the lower extremities. The roentgenogram of the chest was normal. Surgical exploration revealed narrowing of the abdominal aorta at the origin of the renal arteries. The aorta below this point narrowed to the size of the iliac artery. Numerous distended collateral vessels were observed at operation.

Three other cases of the coarctation of the abdominal aorta have been described, but clinical data were not given.[1,10,12]

CLINICAL FEATURES

The symptoms most frequently encountered in cases of coarctation of the abdominal aorta are those associated with hypertension, heart failure, and circulatory insufficiency in the lower extremities. Palpitation, dyspnea, dizziness, headaches, nausea, precordial pain, and weakness and numbness in the lower extremities are the most common complaints. All of these symptoms may also be present in association with coarctation of the thoracic aorta, and they offer little aid in establishing the site of the lesion.

The findings of hypertension of the upper extremities and hypotension of the lower extremities indicate an alteration in the lumen of the aorta or its great vessel trunks, and is present in all cases irrespective of the location. A comparison of blood pressure determinations of the upper and lower extremities may readily establish the diagnosis and will often save the patient

the expense and trouble of irrelevant diagnostic procedures. Palpation of the femoral pulses will often help to establish the diagnosis of coarctation of the aorta, but may be misleading.

Systolic murmurs were present in almost all the cases reported and were loudest just above the area of constriction. The point of maximum intensity was usually in the lower dorsal region posteriorly and near the xiphoid process of the sternum anteriorly, In one case, the murmur was heard as low as the symphysis pubis.

A well established and clinically detectable collateral circulation was present in almost all of the cases. Maycock[7] described three different anatomic pathways in a patient, whose lesion was just below the renal arteries: (1) the seventh, eighth, and ninth intercostals and the internal mammary arteries; (2) the internal mammary and the epigastric arteries; and (3) the subscapular and the intercostal arteries. It is probable that the evidence of collaterals would be detected more frequently with closer clinical observation.

Poor physical development may be noted in the lower extremities. Weakness and pain are common and most likely represent manifestations of circulatory insufficiency.

ROENTGEN FEATURES

The routine roentgen studies of the chest are usually of little aid in the diagnosis of coarctation of the abdominal aorta. Characteristic notching of the ribs was reported in only 1 patient. Widening of the aortic arch was present in 1 case, and another demonstrated an enlarged left ventricle with passive hyperemia of the lungs.

An extensive collateral circulation has been described in almost all of the cases of coarctation of the abdominal aorta; however, notching of the ribs has been reported only once. It is possible that this finding may have been overlooked, or that satisfactory roentgenograms for the demonstration of this phenomenon may not have been obtained.

A method for translumbar aortography was originally described by dos Santos and co-workers[4] and has been revised by Wagner.[11] This has provided a means of positive diagnosis and localization of coarctation of the abdominal aorta. Bahnson, *et al.*[2] employing the usual angiocardiographic techniques, obtained excellent visualization in their case. Both procedures are accompanied by some risk, but to date, serious complications have been rare.

REPORT OF A CASE

A thirty-two year old white man was referred to the North Carolina Baptist Hospital on April 20, 1954, because of nervousness, headaches, palpitation and hypertension. These symptoms had been present for several years, but had become more intense six weeks prior to admission. Hypertension was observed in 1942 during a routine physical examination for the Army. At the age of seven he had received a severe blow in the abdomen in an automobile accident, but recovered spontaneously without sequelae. His physician heard a "heart murmur" at the age of nine years and advised restriction of activities. There was no record of previous renal disease, edema, or dyspnea.

Physical examination revealed the blood pressure in the left arm to be 190 systolic, 105 diastolic, while that in the left leg was 130 systolic, 95 diastolic. The pulse rate was 86 per minute, respiration 18 per minute. The ocular fundi demonstrated marked arteriolar spasm, but no hemorrhages, exudate or papilledema were present. The chest was clear to percussion and auscultation. There was no cardiac enlargement and the rhythm was regular. A systolic murmur was heard in the mid-epigastric area and over the precordium. Posteriorly it was easily heard from the lower dorsal spine to the sacrum. No collateral circulation was observed. The pulsations of the dorsalis pedis and posterior tibial vessels were palpable bilaterally, but were diminished in the femoral artery. Physical development of the lower extremities was normal.

Accessory clinical studies included a normal urinalysis and normal hematologic studies. The blood urea nitrogen was 9 mg. per 100 cc. Serologic tests for syphilis were negative. An abdominal aortogram revealed localized nar-

Fig. 1. Aortogram demonstrating moderate narrowing of the abdominal aorta at and above the origin of the celiac artery.

rowing of the abdominal aorta, with the site of greatest constriction at and just below the origin of the celiac artery.

The usual aortographic technique was used. A long needle was inserted into the abdominal aorta at the level of the twelfth dorsal vertebra, and fortunately was placed just above the site of coarctation. Roentgenograms were obtained immediately after the injection of 20 cc. of 70 per cent diodrast. The concentration of the opaque material was quite satisfactory to demonstrate the lesion, but the collateral circulation was not adequately visualized (Fig. 1).

THEORIES OF ETIOLOGY

The origin of coarctation of the abdominal aorta remains obscure. Maycock[7] suggested two possible etiologies: (1) the failure of the two dorsal aortas to fuse equally at one point, with consequent obliteration or loss of one of them, and (2) kinking of the fused aortas, with the consequent localized increase in longitudinal tension producing a permanent narrowing. Bahnson and co-workers[2] believe that in their case the stenosis was probably of long duration and congenital in origin, although the abrupt onset of symptoms related to deficient circulation in the lower extremities suggested thrombosis of the narrowed segment of the aorta.

In our case, the abnormality had probably existed since the age of nine years. Two years prior to this time the patient had received a severe blow to the abdomen in an accident; the exact relationship of this incident to the present abnormality is a matter of conjecture.

PATHOLOGIC FINDINGS

Six of the cases observed at operation or autopsy revealed the coarctation at either the origin of the renal arteries or just below this point. The coarctation was either a short valve-like structure or a stenosed segment varying from 1 to 3 cm. in length. The lumen was reduced in diameter, and in several cases was occluded by a mass—either a blood clot or a calcified plaque. In most instances an extensive collateral circulation was demonstrated. The aorta proximal to the stenosis frequently revealed moderate to marked thickening of the entire wall, marked arteriosclerosis, and thickening of the intima with ulceration. The aorta immediately above the coarctation was normal or dilated. Aneurysm formation was also described. The segment distal to the coarctation was usually reduced in caliber and failed to reveal any dilatation, thickening, arteriosclerosis, or aneurysm formation. It appeared to be patent in all cases.

DIAGNOSIS

Coarctation of the abdominal aorta produces symptom similar to those found when the aortic arch is the site of the lesion. The diagnosis of coarctation is easily established by the finding of hypertension in the upper extremities and hypotension in the lower extremities. Pulsations in the femoral arteries may still be palpable when coarcta-

tion of the abdominal aorta is present without complete obstruction. Hence, without accurate blood pressure determinations in both upper and lower extremities, the diagnosis may be missed. The collateral circulation and the systolic murmur found in the lower dorsal and upper lumbar regions may often be the only clinical findings which are helpful in determining the location of the lesion.

The routine roentgenographic studies offer little aid in the diagnosis. The chest roentgenogram is usually normal, but may possibly demonstrate notching of the lower ribs, cardiac enlargement, and pulmonary passive hyperemia.

Roentgen evidence of arteriosclerosis distal to the lesion is more indicative of obstruction due to thrombosis rather than to coarctation. The aorta is best visualized by injecting an opaque substance directly into the lumen. Serial film studies following such an injection may demonstrate the collateral pathways.

TREATMENT

Surgical correction of coarctation of the abdominal aorta has been attempted on one occasion. Sympathectomy and utilization of the omentum as a potential source of collateral circulation have been tried without success.[2] The usual location of the lesion at or near the origin of the renal arteries and the superior mesenteric artery has made the surgical approach to this condition extremely difficult and hazardous. Perhaps the ingenuity of the surgeon will make operative correction possible.

SUMMARY

1. A case of coarctation of the abdominal aorta is described and the literaure concerning this subject is reviewed.

2. The value of aortography in the diagnosis and localization of this lesion is emphasized.

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Vol. 76, No. 4

TECHNIQUE OF SERIAL LONG FILM ANGIOGRAPHY IN ARTERIOSCLEROSIS OBLITERANS*

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THE direct operative attack upon arteriosclerosis represents one of the most challenging problems in modern day surgery. Increasing numbers of pilot studies all over the world are testing the value of thrombendarterectomy and graft replacement procedures as methods of definitive treatment for obliterative disease of the aorta and major peripheral vessels. It is frankly questionable whether accurate diagnostic tests have yet been perfected for proper case selection, and one wonders whether these therapeutic trials are running ahead of clinical rationale (Cranley, Rob). Whatever may be the judgment of time, it is clear that vascular surgeons are turning with renewed interest toward the dismal rows of beds that have in the past been reserved for geriatric amputees (see references to the work of Barker, Bogardus, Cooley, Cranley, de Takats, Deterling, Fontaine, Herrmann, Hufnagel, Julian, Linton, Oudot, Rob, Self, Szilagyi and Wylie).

The radiologist is the surgeon's servant, helpmate, colleague and conscience. He is obliged not only to keep abreast of the need for new roentgenographic services but to take care in evaluating new methods critically. Angiography is a case in point. Experience has already shown that no preoperative patient with vascular insufficiency can be considered adequately evaluated until he has had, among other studies, a "suitable" angiographic examination. The word "suitable" implies to this author a conservative balance among the surgeon's need for information about the patient's vessels, the radiologist's interest in good roentgenograms, and the patient's right to comfortable survival following any diagnostic procedure.

The long-film angiographic techniques described in this paper have been employed at this institution since March, 1953, and represent a frank compromise among the factors mentioned above, as well as cost and convenience factors. Since it is not claimed that they are ideal, it seems important to preface their description with allusion to other workers' splendid achievements in producing long film angiograms. A general history of aortography and femoral arteriography is inserted to help place these techniques in historical perspective.

HISTORY OF AORTOGRAPHY AND FEMORAL ARTERIOGRAPHY

The clinical use of opaque substances for arteriography was born with Brooks'[13] experiments with sodium iodide, reported in 1924. Berberich and Hirsch[8] had employed strontium bromide earlier, and Sicard and Forestier[73] made attempts with lipiodol, but neither of these substances proved acceptable.

Credit for the adoption of arteriography as a routine clinical procedure, however, clearly lies with dos Santos. Stimulated by the studies of cerebral arterial circulation begun in 1927 by Egas Moniz and coworkers,[27,28] dos Santos, *et al.*[68,69,70] in 1929 employed sodium iodide for aortography and femoral arteriography. He is unequivocally the pioneer in aortography, publishing his historic book in 1931.[71]

In that same year, Pearse and Warren[57] at this hospital may have been the first to report the use of an organic iodide compound (skiodan) for femoral arteriography. By 1935 Reboul[60] had considerable experience with both procedures. Sporadically during the 1930's, various North American authors reported series of peripheral ar-

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teriograms, mainly employing thorotrast.[1,2,25,26,80,86]

Aortography was not reported in the United States until 1942, when Doss, *et al.*[23] and Nelson[52] commented on its value in diagnostic problems of the abdominal viscera. Actually, Henline and Moore[36] had become interested in its possibilities as far back as 1936, having studied dos Santos' and Osorio's[53] work. Employing various opaque substances, particularly sodium iodide, their preliminary experiments in dogs had been disastrous, and they interpreted them as a signal to abandon any attempt at aortography in humans. (dos Santos had already warned that dogs were not suitable comparative subjects for aortography.) Added to this failure was a widely publicized misinterpretation or mistranslation of Balestra's[4] comments concerning his observations in dos Santos' clinic. As a result, translumbar aortography was discouraged in this country until 1942. By that time the war interrupted the dissemination of the technique in the United States, although Europeans and South Americans continued to gain experience in both aortography and peripheral arteriography.

Even after the war, aortography was not generally discussed in North American journals until reports by Wagner and coworkers[81,82] were published in 1946 and 1947, and by Melick and Vitt[49] in 1948. Despite the large backlog of experience accumulated in Europe during the 1930's and 1940's, it was really Smith and his urologic colleagues[74] in Cincinnati who succeeded in placing diagnostic aortic puncture on a "respectable" footing in this country. By February, 1952, their renal angiogram series included 1,000 cases without a fatality. Translumbar aortography rapidly developed a significant place in urologic diagnosis in this country.

The use of aortography by vascular surgeons has had a parallel but somewhat delayed development in this country. Even femoral arteriography had been abandoned after its early development here. Except in isolated cases of arteriovenous fistula, embolism or trauma, such examinations appeared fruitless and even contraindicated in the view of many North American surgeons.[9] A large reservoir of surgical problems faced them in their arteriosclerotic patients, but conservative medical opinion awaited some therapeutic justification for these diagnostic procedures.

With the evolution of surgical techniques aimed at correcting arteriosclerotic lesions, this justification rapidly reached both surgeons and radiologists. Some of the proposed operations are simple in principle, such as Cid dos Santos' original thromb-endarterectomy.[65,66] The more complex grafting procedures can be astonishingly dramatic and—what is more important—gratifyingly successful. Paralleling this intensive surgical research, the practice of aortography and femoral arteriography has become a necessity in every medical center that has an active vascular surgeon.

LONG FILM TECHNIQUES

Serialization. At the outset it is important to stress the preference for serialized exposures, a subject which has engaged foreign workers for years (see numerous references in following sections). Perusal of the illustrations and legends following within this paper will demonstrate to the reader how quickly this necessity was impressed upon the author. A single roentgenogram, *in a relatively normal patient,* can demonstrate successfully the aorta and peripheral arteries simultaneously after rapid injection. However, sluggish circulation due to long or multiple segmental blocks and arteriolar spasm are so common in the type of patient under study that multiple films seem obligatory if one expects to obtain a complete anatomic demonstration. There are, theoretically, techniques of slow or intermittent injection which possibly circumvent this need, but they require increased dosages of opaque substance with increased risk to the patient.

If a timing clock image[1] is projected upon

[1] The only such clock which I know to be available commercially is that described by Fullenlove.[31] Bonte and Cordier[12] and Thoyer-Rozat, *et al.*[78] show clock images on their roentgenograms. Dr. Paul Hodges developed one for angiography at the University of Chicago. Mr. James Rains has made a clock for us locally.

each serial roentgenogram, the examination may provide additional interesting physiologic data concerning the circulation.

Femoral Arteriography. Specifically pertinent to this paper is a review of literature in which aortographic and femoral arteriographic techniques employing long films and covering wide areas have been described. Although numerous ingenious methods have been devised for study of the femoral artery, those that permit the making of only a single roentgenogram of the entire extremity will not be dealt with in detail.[3,6,21,22,34,40,47] More important are those which provide for serial long roentgenograms of one entire extremity,[7,16,32,39,43,51,55,56,63,75,83] the epitome of which is the technique of Bonte, *et al.*[11,12] He has constructed a long hexagonal drum which holds six cassettes, each approximately 5½ inches wide by 39 inches long. The drum is rotated by a hand crank at one end, permitting exposures at three second intervals. The entire apparatus can be turned upright for venography of the leg. Ducuing and his associates[24] have adopted the same equipment.

Simultaneous Aortography and Femoral Arteriography.

A. *Two Injections.* Many individuals have noted the possibility of visualizing the aorta and circulation of both lower extremities following aortic injection, and have developed techniques of varying usefulness. In 1948, Leroux[44] discussed a technique which is still being employed in clinics where long cassettes are not available. After one aortic injection, a single roentgenogram is made of the abdomen and pelvis; then a second injection is performed and, after waiting a few seconds, another long composite roentgenogram is made of both extremities. These two roentgenograms, placed end-to-end, produce a static view of the arterial anatomy from the point of aortic puncture down through the ankles.

B. *Single Injection.* Methods for depicting the circulation of the lower abdomen, pelvis and both lower extremities after a single aortic injection have also been described. Wagner, *et al.* [82] in 1947, suggested placing two 14 by 17 inch cassettes end-to-end under the patient, exposing the abdominal film with one tube just at the end of injection, and exposing the film under the thighs shortly thereafter with a portable machine positioned over the lower portion of the field. Dos Santos[67] once suggested a similar two-tube method, and individuals in this country have been using it. Gottlob, *et al.*[33] exposes one film under the aorta and iliac vessels; a preliminary fluorescein study of arterial circulation time then dictates the optimum moment for exposing another cassette under thighs and legs by means of a second tube. Pereira-Caldas has been using a two-tube method since 1942.[58,59]

Christmann and Grinfeld[14] employ a small film changer. After a single aortic injection of 50 cc. of opaque substance, the patient is pulled up the table in stages so that different levels may be filmed as the opaque material descends into the legs. This is ingenious but of doubtful dependability, and does not represent either long filming or serialization.

It is much less complicated to employ a long film to begin with, the entire length exposed by means of a single roentgen tube. The only requisite for this method is a tube mounting which permits adequate target-film distance for long film coverage. And it is desirable to provide for serialized exposures.

Since 1948, Bazy, *et al.*,[7] Reboul and Laubry,[61] and Thoyer-Rozat and co-workers[79] have been employing a film changer which can take six serial roentgenograms, each 8 by 47 inches, or three serial roentgenograms of size 16 by 47 inches. Because details of its construction are not published, it is not known whether one or more roentgen tubes are employed. Gobin and Duvezin[32] diagrammed a different and less promising type of device for making serial roentgenograms of the same size. An apparatus designed by Robert[63] can be employed to make three 14 by 34 inch roentgenograms. It is obvious that Moeys and Van der Valk,[51] and Bätzner and

Bracht,[6] Loose[48] and De Reus and Vink[18] have been accomplishing similar serialization of long, wide films, probably for several years.

Pereira-Caldas claimed at one time (1952) that he was developing a serial long film device for combined aortography and femoral arteriography with only one injection, but this has not yet been described.

In this country, Wylie and McGuinness[85] in 1953 were the first to describe serial long film aorto-arteriograms. According to a personal communication from Miller,[50] they use a simple film tunnel to transmit four 14 by 36 inch cassettes, the tunnel being surfaced by two 14 by 17 inch Lysholm wafer grids, end-to-end. The cassettes are placed on a small table at one side of the patient and are pushed through the tunnel across the axis of the roentgen table where they are caught by an assistant after each exposure. Following one injection of 50 cc. of 70 per cent urokon, four 14 by 36 inch cassettes can be pushed through the tunnel in eight or ten seconds. These produce serial images of the aorta, the pelvic vessels and the arteries of both extremities down to the level of the popliteal bifurcations. Langsam, *et al.*[42] have more recently described their own 14 by 36 inch film tunnel.

The present author[64] has been employing serialization with mixed 14 by 36 inch and 14 by 17 inch cassettes since early in 1953. However, general availability of this size cassettes in this country, owing to chiropractic demands, had encouraged several others to employ them for aorto-arteriography at about the same time, usually for single exposures.[29,35,76] Several European authors also report the use of single long films.

The only serial long filming device commercially available at this time is the modification of Gidlund's machine produced by Elema-Jarnh, Ltd., Stockholm, Sweden. Model DST-655 is said to produce serial 14 by 32 inch films at a rate varying from one per ten seconds, up to three per two seconds.

AUTHOR'S METHODS

I. *Serial Long Film Aorto-arteriography.* Between March, 1953, and July, 1956, 125 long film aorto-arteriograms were completed satisfactorily at this hospital by a compromise method which yielded satisfactory diagnostic arteriographic information from the level of the midlumbar aorta down to the bifurcations of both popliteal arteries. Several additional attempts were classified as failures because of improper aortic puncture.

A. *Roentgen Equipment.* The table is equipped with a hand-cocking 14 by 36 inch Potter-Bucky diaphragm and tray[2] permanently mounted and in routine use for other examinations also. The mechanical and grid characteristics of our particular diaphragm require at least a 0.3 second exposure time. Target-film distance of 52 inches allows exposure of the film throughout its 36 inch length. The roentgen tube is aligned so that its long axis is across the table to avoid the "heel effect." Density variations from the abdomen down to the calf are then compensated for by the use of an aluminum filter 4 mm. thick combined with a sheet of 18-gauge stainless steel, placed under the caudal half of the tube aperture. (Several workers deliberately employ the "heel effect" and accentuate it by angling the tube caudad. Others have improvised wedge filters.)

Two sets of films in cassettes are employed as follows: (1) Two 14 by 36 inch cassettes equipped with average speed screens, loaded either with single sheets of 14 by 36 inch screen-type roentgen film, or with two 14 by 17 inch films placed end-to-end inside the cassette and (2) four 14 by 17 inch cassettes with similar screens and films for roentgenographing the lower half of the field late in the examination.

We began this work originally with only one long cassette for the first exposure, plus additional 14 by 17 inch units for all

[2] Potter-Bucky grid, cassettes and film are commercially available through local x-ray supply houses throughout the United States.

subsequent exposures. In one case of long iliofemoral block, the exact route and level of femoral artery reconstitution could not be appreciated by studying the first two exposures. As a result of this experience, we invested in another long cassette in order to cover the entire area at least twice early in the examination (Fig. 1).

B. *Positioning, Puncture and Injection.* The patient lies prone on the table, most frequently under spinal analgesia or sodium pentothal anesthesia. A preliminary survey roentgenogram is studied by both surgeon and radiologist in order to decide the best point and angle of puncture so that the needle will enter the aorta in a cephalad or countercurrent direction, well below the presumed levels of the renal arteries. A 17-gauge styletted needle of thin flexible steel, with lumen equivalent to the usual 16-gauge,[3] is then inserted percutaneously into the aorta through the left lumbar region. After the aorta has been punctured, a non-expansible plastic tubing with Luer-Lok adapters is attached to the needle. (It is advisable to check the needle position by making a test exposure after injection of 5 to 10 cc. of opaque material; however, this has not been done in many of our cases.) Using an ordinary 30 or 50 cc. glass syringe with needle lock, 30 to 40 cc. of 70 per cent urokon is injected by hand as smoothly as possible in approximately three or four seconds.

C. *Exposure Timing and Film Changing.* The first exposure is made during the injection of the last 5 cc. of contrast medium to insure the presence of a continuous column of opaque substance in the aorta. Two practiced assistants are needed to change the cassettes in a minimum of four seconds. One pulls out the tray and removes the exposed cassette. The second immediately inserts an unexposed cassette, pushes in the tray, and cocks the Potter-Bucky diaphragm. A third person operates the exposure button.

FIG. 1. (*1*) Aortogram: value of at least two consecutive long films early in the examination. First 14 by 36 inch film reveals long block of common iliac, external iliac and common femoral arteries on the patient's left.

As mentioned above, the first two exposures cover the entire 14 by 36 inch area. Subsequently, four 14 by 17 inch cassettes, placed in the lower end of the

[3] This needle was supplied in 1954 by the Becton-Dickinson Company, Rutherford, New Jersey (Stock Number: T-466-LNRH, 17-gauge, 7-inch). However, all of the illustrated aortograms were done earlier, using a standard 18-gauge needle and only 30 cc. of 70 per cent urokon.

tray, are exposed to visualize the opaque substance around the knee and below if its descent is delayed by any anatomic or physiologic abnormality (Fig. 2 and 3). The long cassettes and the first two subsequent exposures are changed as rapidly as possible. The fifth and sixth cassettes are exposed leisurely over a period of approximately one minute after the start of injection, in the hope of demonstrating the veins of the leg, if possible.

A portable roentgen-ray machine centered over the lower legs and feet may be employed to expose a single 11 by 14 inch cassette placed on the tabletop.

D. *Exposure Factors.* At the 52 inch target-film distance, exposure factors for an average patient are approximately 70 to 75 kv., 200 ma. at 0.5 second.

Fig. 1. (*2*) Second exposure, employing only a 14 by 17 inch film under thighs, indicates that the superficial femoral artery is indeed patent, but does not show the exact route or point of reconstitution higher in the thigh (presumably by refluence from deep femoral artery). This case stimulated the purchase of a second 14 by 36 inch cassette. Also note completely blocked short segment of the right common femoral artery below the inguinal ligament and an incomplete block in the proximal right superficial femoral artery.

E. *Area Examined.* In the average patient, the 14 by 36 inch roentgenogram permits visualization of the area between that portion of the aorta opposite the second lumbar vertebral body and the points of branching of the peroneal arteries from posterior tibial arteries in both legs (Fig. 4).

II. *Serial Femoral Arteriography.* Prior to the adoption of this aorto-arteriographic technique, we were asked to devise a procedure for serial femoral arteriography. It has proved most useful on occasion, and it is described here for those readers who may need or prefer the femoral approach.

A. *Roentgen Equipment.* The 14 by 36 inch Potter-Bucky diaphragm is again employed under the tabletop as described for the aortographic technique. However, the film tray is removed and replaced by a sheetmetal copy which does not have the usual cassette locks. The tabletop is masked with sheets of lead $\frac{1}{8}$ inch thick except for an opening 7 by 36 inches in the center of the table over which the thigh and leg are positioned.

A target-film distance of 52 inches is again employed with the same equalizing filter in the tube aperture as described for aortography.

Two 14 by 36 inch cardboard filmholders are placed in the tray, one on top of the other. Lead backing, $\frac{1}{16}$ inch thick, is incorporated inside the upper holder. This serves to protect the bottom film during the two initial exposures. Each filmholder is loaded with a sheet of 14 by 36 inch non-screen film or with two 14 by 17 inch non-screen films placed end-to-end. The cardboard holders are placed eccentrically toward the outer margin of the tray in position for the first exposure along one 7 by 36 inch half of the top film (Fig. 5).

B. *Positioning, Puncture and Injection.* The patient lies supine on the roentgen table, under anesthesia or analgesia. The

FIG. 2. Aortogram: need for serialization. (*1*) First long film (left) does not demonstrate the nature of superficial femoral artery block in the patient's left thigh. This is due to sluggish circulation, and not to femoral spasm. (*2*) Second long film (right), approximately four seconds later, shows the blocked segment to be short and demonstrates the state of the popliteal artery and its branches as far as the film length permits. Note partial nephrogram, right kidney via anomalous renal artery, despite unusually low point of injection. No gross adverse clinical effect.

Fig. 3. Aortogram: value of serial filming. (*1*) The first long film (left) reveals block of the left common iliac artery at A; reconstitution at B, but block of both superficial femoral arteries at points C. (*2*) Not until the second film (14 by 17 inches, upper right) is the reconstitution of both superficial femoral arteries appreciated at points D. (*3*) Additional detail at and below the knee follows in third exposure (lower right). No gross clinical sequelae from flooding renal artery in this case.

FIG. 4. (*1*) Aortogram: area covered by 14 by 36 inch cassette in average patient. (*2*) Note that later serial 14 by 17 inch films over at least a ten second period (right) are required to demonstrate fully the state of arteries below the knee. (*3*) Even a more delayed exposure was successful in visualizing the popliteal branches on the patient's right, since circulation in this leg is slowed by external iliac and common femoral block as well as by general vasospasm.

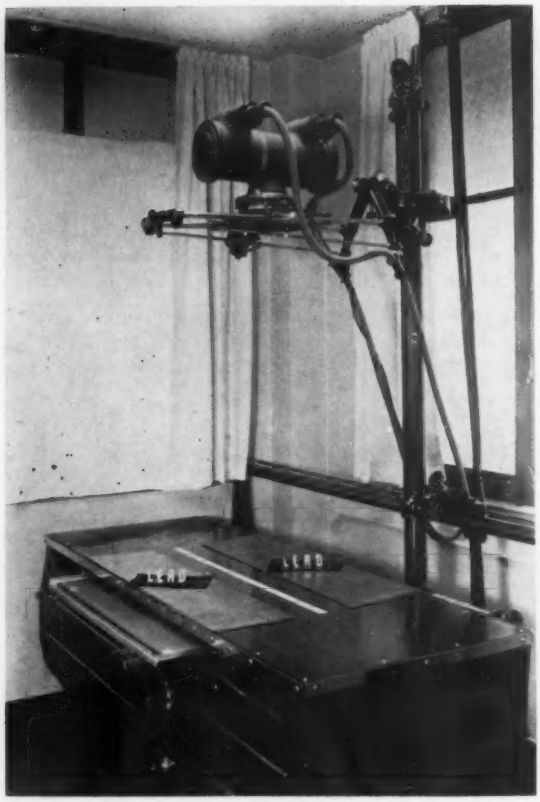

Fig. 5. Femoral arteriography technique. Two 14 by 36 inch cardboard holders stacked eccentrically in Bucky tray. One eighth inch lead masking leaves 7 by 36 inch space on tabletop for filming the extremity twice with each long filmholder.

leg is extended, preferably in internal rotation to permit projection of the popliteal artery branching into the space between fibula and tibia. Compression bands across knee and foot aid in maintaining position.

An 18-gauge needle with short bevel is inserted percutaneously into the common femoral artery just below the inguinal ligament. It is pointed cephalad against the direction of blood flow. The syringe and plastic tubing are attached in the same manner described above for aortography. It is well to have an assistant steady the needle with a clamp during injection.

The opaque medium is a 25 cc. bolus of either 50 per cent diodrast or 50 per cent urokon, and it is injected manually within a maximum of six seconds. Following the suggestion of many experienced workers, a lower concentration of opaque medium and slower rate of injection than used during aortography are employed during femoral arteriography, in order to minimize the possibility of vasospasm. We have even obtained readable roentgenograms with 30 or 35 per cent concentrations.

C. *Exposure Timing and Film Changing.* The first exposure is made during the injection of the last 5 cc. of bolus. A technician stands by with an improvised cue stick and rams the top filmholder across the tray to prepare for the second exposure along its unexposed half (Fig. 6). Following the second exposure, the top filmholder is pulled out of the tray by means of adhesive or gauze strapping, leaving the bottom filmholder in position for third and fourth exposures in a manner similar to the first and second (Fig. 7). Thus, four sequential exposures may be obtained at minimum intervals of about two seconds, although it is preferable usually to prolong roentgenography over a period of approximately 12 or 15 seconds.

D. *Exposure Factors.* Non-screen film with Bucky: 100 kv., 200 ma. for 1.0 second. The roentgen tube position and filtra-

Fig. 6. Femoral arteriography, Bucky tray detail. After exposing one long half of top film, technician rams filmholder across the tray with stick, preparing for second exposure. Masking is done on tabletop (see Figure 5).

tion are similar to those described above for aortography.

E. *Area Examined.* In an average adult male patient, this is from the inguinal level down the leg to a point just above or at the ankle. In a short patient, the foot is frequently included (Fig. 8).

F. *Variation with Screen Cassettes.* The use of non-screen film permits stacking a full complement of film within the tray in two cardboard holders. However, in thicker patients the non-screen roentgenograms tend to appear gray and to lack contrast. In such cases, 14 by 36 inch screen cassettes may be employed with screen film. Two exposures of the first cassette are made by the method of eccentric film advancing just described; whereupon the first cassette is removed rapidly and another inserted for third and fourth exposures. The following are average technical factors: 60 to 65 kv., 200 ma. for 0.5 second.

III. *Venography.* The techniques described above apply equally well for venographic examination of the lower extremity. We are able to obtain the necessary target-film distance with the table either horizontal or in a 45-degree upright position.

FIG. 7. Femoral arteriography. After two exposures of top film, it is pulled from tray by gauze strapping. Bottom film is then ready for exposures 3 and 4.

FIG. 8. Femoral arteriogram. (*1* and *2*) First two exposures made in a single 14 by 36 inch cardboard holder. Note excellent filling of external iliac artery by refluence. Later films showed good venous return detail, including common iliac vein. In this instance, needle has inadvertently been inserted into the origin of the deep femoral artery, but countercurrent injection is successful. Assistant steadies needle with clamp during injection of 25 cc. of 50 per cent urokon.

SUMMARY

1. The general history of aortography and femoral arteriography is reviewed, with particular attention to techniques covering long, wide areas.

2. The importance of serialized exposures is stressed in the study of patients with arteriosclerosis obliterans.

3. A method of serial long film aorto-arteriography is described, employed since March, 1953 in the study of arteriosclerosis at the Strong Memorial Hospital, Rochester, New York.

This involves the use of a 14 by 36 inch Potter-Bucky diaphragm installed under the tabletop; 14 by 36 inch and 14 by 17 inch cassettes; low translumbar puncture of the aorta and injection of 70 per cent urokon.

4. A method is also described for serial femoral arteriography by percutaneous puncture, employing the same Potter-Bucky diaphragm and 14 by 36 inch cardboard filmholders loaded with non-screen film.

5. The serial femoral method is also adaptable to venography in either erect or horizontal positions.

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The author wishes to express his very great appreciation to the following individuals for their help in this project, and with other aspects of this subject to be discussed in additional papers: W. J. Merle Scott, M.D., Professor of Surgery and Surgeon-in-Chief, for providing the clinical stimulus and senior guidance, and for referring most of the patients at the outset; Messrs. John Hollenbach, William Otto, Richard Wolfe, James Rains and William Williams for invaluable technical assistance, Glenn E. Jones, M.D., for suggestions concerning film transport in femoral arteriography; William S. Cornwell, Associate in Radiology, for editorial assistance; and George H. Ramsey, M.D., Professor of Radiology and Radiologist-in-Chief, for his encouragement, mature guidance and counsel.

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A DEVICE FOR RAPID SERIAL ANGIOCARDIOGRAPHY IN THE ERECT POSITION

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THE burdens placed upon a busy roentgenologic service by time-consuming special procedures such as angiocardiography dictate the need for simplicity of apparatus, rapid but accurate positioning of the patient, and easy film processing.

The requirments for good angiocardiography as outlined by Morgan,[1] and more recently by Wyman and Scholz,[3] are briefly listed as follows:

1. Simplicity of apparatus, to avoid mechanical failures.
2. Films of adequate size and of excellent quality.
3. Rapid sequence of films with readily adjustable speed combinations.
4. Shielding of personnel against radiation in excess of minimum safe limits.

These requirements are believed to be fulfilled for the erect position by the apparatus now used by the authors. The apparatus consists of a simple, gravity-drop automatic unit employing conventional 14 by 17 inch cassettes. The erect positon, with the patient seated in front of the angiocardiographic unit, was decided upon for several reasons. First, it allows films to be exposed in the conventional posteroanterior erect position, and comparisons with plain films may easily be made. Second, circulatory hemodynamics appear to operate more favorably in this position, that is, the bolus of contrast substance remains well concentrated. This may be due to the ability of the patient to make a better sudden inspiratory effort in the erect position. It has been our experience that at least 90 per cent of the adult patients referred for study can easily tolerate the injection while in the sitting position.

The large size film was decided upon because it insures rapid and accurate positioning of the patient, and vessels not included on smaller films are easily identified. The latter is of some importance in cases demonstrating opacification of transverse cervical, carotid, or axillary arteries in the soft tissue at a time when definite evidence of aortic visualization is otherwise lacking. Again, unsuspected venous anomalies of the systemic or pulmonary circulation are often encountered.

Gravity-drop methods have been employed previously for rapid serial angiography. An ingenious device, utilizing the principle of the inclined plane, was described by Sussman and Grishman.[2] This unit held five cassettes, each 10 by 12 inches in size. The interval between exposures was not stated.

The present apparatus consists of four 21 inch long brass worm shafts, placed horizontally at the sides of the top and bottom of the upper compartment. The space between the threads (1 inch) is slightly larger than the thickness of a conventional 14 by 17 inch cassette. Space was allowed for 18 cassettes to be exposed at the rate of two per second. The four shafts are fitted with sprocket gears and connected by a link chain attached to the drive shaft of a 1/12 H. P. motor through suitable gear reduction. As the worm shafts turn synchronously, the forward pitch of the threads advances the cassettes which ride in the upright position on steel runners placed at the sides of the compartment bottom.

The exposures are made by means of a microswitch placed centrally in the floor of the compartment near the end of the steel runners. This switch is depressed by the bottom edge of each cassette before it begins its vertical fall. A stationary wafer

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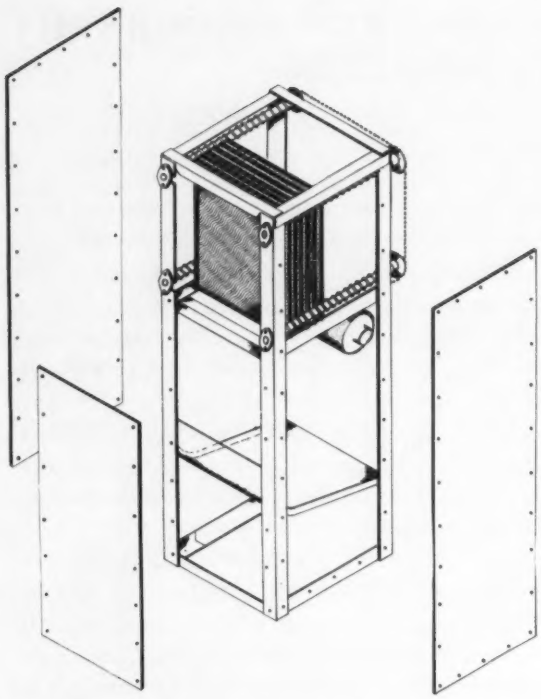

FIG. 1. Line drawing of the unit with side and front panels removed.

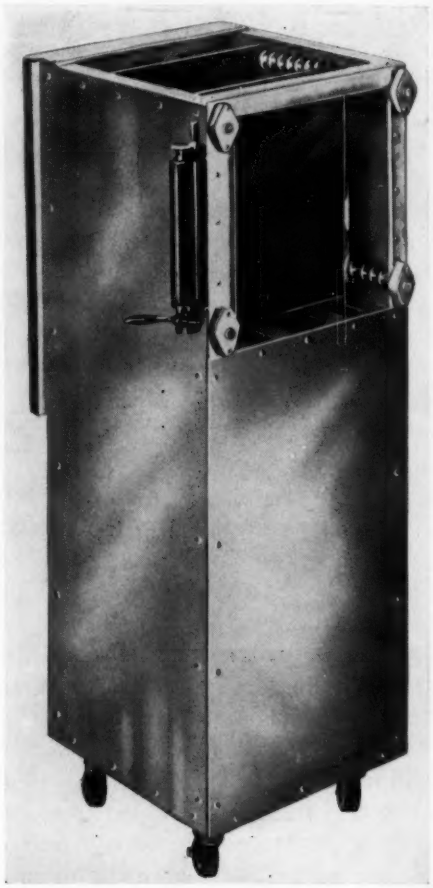

FIG. 2. The vertical cassette changer with stationary wafer grid removed.

grid is mounted on the frame at the front of the upper compartment. The cassettes are backed with a layer of 1/32 inch thickness of lead placed behind the back intensifying screen. The lead filters supplied for industrial million volt roentgenography are available in this thickness and in convenient 14 by 17 inch size. Although the forward motion of the cassette continues during the exposure, it does not effect the quality of the film. Since all motion is in the direction of the axis of the roentgen beam during the exposure, there is no blurring of the image. The cassettes fall vertically upon a curved chute placed in the bottom compartment. Their motion is thus translated to the horizontal plane and they are collected in a cushioned box placed at the back of the unit.

At present, the gear ratio is established to provide an exposure rate of two films per second. If it is desirable to limit the rate to one film per second, the cassettes may be placed at alternate spaces in the threaded worm gears. Since there are 18 spaces, this would result in a maximum of 9 cassettes at the rate of one per second. There is an advantage in this arrangement, inasmuch as the rate of exposure may be varied during the study. For example, if it is desirable to visualize right heart opacification at the rate of two per second, and the left heart at one per second, cassettes may be placed at each available space for a total of 8 to 10 and the remainder in alternate spaces. In this manner, the radiation hazard is reduced and roentgen tube life and films are conserved.

The patient is seated upon an adjustable clinic stool in front of the unit. For the posteroanterior projection, the patient's knees must straddle the lower compartment. This position is somewhat strained, but it is not difficult to maintain for the

duration of the examination. A compression band is used for immobilization.

The entire unit is mounted on rubber-tired casters for mobility. At present, blocks are used to raise the casters above the floor level for stability during operation. The dimensions of the compact unit are as follows: height 48 inches, width 20 inches, depth 23 inches. The unit approximates the size of a standard steel filing cabinet and may be used as a convenient cassette support for routine chest films in any room of the roentgenologic department.

The apparatus may also be used for those patients who are not able to tolerate the procedure in the upright position, and for abdominal aortography, provided there is no contraindication to the lateral decubitus position. In this event the unit is brought to the side of the roentgenographic table and the tube is directed horizontally. Lateral films may be easily exposed with the patient in the supine position.

For posteroanterior projection, it is necessary that the patient assume the lateral decubitus positon.

SUMMARY

The following are the advantages of this unit:

1. It is simple in construction and operation and, as far as possible, eliminates mechanical failures.

2. The use of 14 by 17 inch film insures quick and accurate positioning, a more complete angiographic study, and easy film processing.

3. The rate of exposure, two per second, covering an elapsed time period of nine seconds with 18 cassettes, compares favorably with existing commercial equipment.

4. The rate of exposure may be varied during the study which results in roentgen tube and film conservation and decreased radiation to the patient and personnel.

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The authors wish to express their gratitude to Lt. Col. Mourice J. Fletcher, M.S.C., Director of the Army Prosthetics Research Laboratory, for valuable assistance in the construction of the unit.

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A TECHNIQUE IN PERCUTANEOUS CEREBRAL ANGIOGRAPHY

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PERCUTANEOUS cerebral angiography has become a routine procedure. It is not difficult to penetrate the common carotid artery. With enough perseverence, this can always be accomplished. However, success depends mainly on the introduction of the entire bevel of the needle inside the lumen of the common carotid artery. If the bevel is partly in the lumen, various complications and failure result. Arterial blood escapes either through the open bevel into the soft tissue of the neck or into the wall of the carotid artery. Similarly, during the actual injection, extravasation of the contrast medium into the soft tissue occurs if part of the bevel is outside the arterial wall. Extravasation of either blood or the contrast medium, particularly the latter, causes vascular spasm locally or distally in the internal carotid artery. This invariably results in unsatisfactory percutaneous cerebral angiography.

Many authors have described the general technique and the successful performance of percutaneous cerebral angiography.[1–8] None has discussed in detail the technique of the most important part of percutaneous cerebral angiography, namely, the threading of the needle into the lumen of the artery.

At Temple University Hospital and Medical School, the Chamberlain bi-plane stereoscopic serial angiographic unit is used exclusively for cerebral angiography.[5] The unit consists of two Fairchild magazines placed at right angles to each other on a circular frame capable of taking twelve pair of consecutive anteroposterior and lateral skull films simultaneously within seven seconds; any consecutive films are a stereo pair. This is achieved by using only one single injection of the contrast medium, usually 10 cc. of 30 per cent sodium urokon solution.[5]

We learned from our experience that three points in the technique of performing satisfactory percutaneous cerebral angiography should be stressed.

1. *Position of the Patient.* The patient's head should be extended as much as possible and moved as little as possible once the needle is introduced into the artery. To obtain a satisfactory anteroposterior view, even with the head in extension, we place our patient on an examining table in a semi-reclining, sitting position with about 60 degree inclination.[5] Even though the roentgen tubes in Chamberlain's angiographic unit always remain perpendicular to the surface of the cassettes, we are still able to obtain a routinely satisfactory anterposterior view midway between Chamberlain-Towne's and Waters' view.

The importance of hyperextension of the neck in order to put the common carotid artery in tension and the avoidance of movement of the head after the needle is introduced in the artery are well known precautions.

2. *The Needle.* We have tried various needles in percutaneous cerebral angiography and have found the ordinary long bevel, 3 inch, 18 gauge needle best. The needle has to be extremely sharp, not only at the tip but, also, along the edge of the bevel. In order to penetrate the artery with ease, one needs not only the piercing effect of the tip of the needle, but, also, the sharp, cutting effect of the edge of the bevel. For each procedure, a brand new needle is used. A re-sharpened needle is never employed. Care also should be taken to protect the tip of the needle during autoclaving. Sometimes, it may be wise to penetrate the tough

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skin of the neck with a separate needle so as not to dull the point of the needle used for the penetration of the artery.

3. *"Snap" of the Artery.* From our experience, this "snap" is the most important guide in threading the needle into the lumen of the common carotid artery. With the left forefinger tip resting on the common carotid artery where it is most strongly pulsating, usually 2 inches above the clavicle, the tip of the needle is first guided to the point between the fingertip and the artery (Fig. 1*A*). A quick thrust is then made, attempting to pierce through the entire artery (Fig. 1*B*). The needle frequently passes through the artery without the operator's knowledge and no blood jetting back into the syringe is noted. This is probably because the two arterial walls are being approximated when the needle is pushed through.

One makes no attempt to thread the needle directly into the lumen of the artery on the initial penetration. When the artery is believed to be pierced, the tip of the needle is slightly elevated toward the surface of the skin, carrying with it the "speared" carotid artery (Fig. 1*C*). With the tip of the needle still gently elevated, one then withdraws the needle gradually. When part of the bevel re-enters the lumen of the artery, arterial blood appears in the syringe (Fig. 1*D*). Withdrawal should be continued slowly until the tip of the needle passes through the deeper wall of the artery. At this instant, a definite snap is felt at the tip of the needle due to the sudden recoil of the uplifted deeper arterial wall (Fig. 1*E*). When this is experienced, the tip of the needle is in the middle of the lumen and the withdrawal should be stopped immediately. The angle of the needle is now lowered toward the surface of the skin and with the bevel of the needle pointed upward, the needle is advanced gently 5 to 10 mm. toward the cranial end (Fig. 1*F*), and this

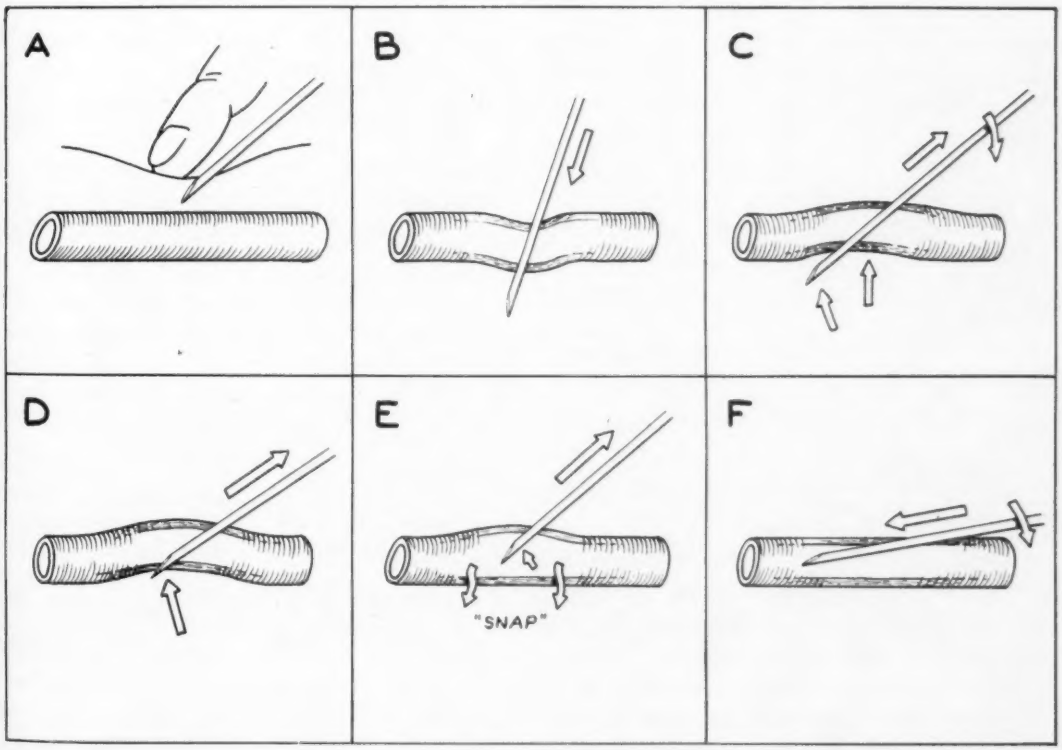

Fig. 1. Diagrammatic illustration of the "snap" of the carotid artery elicited during the percutaneous cerebral angiography.

securely threads the entire bevel of the needle into the lumen of the common carotid artery. Intra-arterial injection of the contrast medium can then be carried out.

Very rarely one misses the snap of the deeper wall, and, instead, experiences the snap of the anterior wall of the common carotid artery. The appearance of venous blood from the jugular vein or the cessation of the arterial blood flow in the syringe would then occur. The procedure must then be repeated.

Since in our setup only one injection is necessary, we simplify the procedure by attaching a 10 to 20 cc. syringe directly to the needle through a two-way stopcork. We believe that it is easier to manipulate and to control the needle with a syringe directly attached and make no attempt to fasten the needle to the skin. The bevel of the needle remains pointed upward during the entire procedure because we find no benefit in altering the position of the bevel during the injection.

The snap of the artery can be felt if the tip of the needle is being elevated during the withdrawal in almost 90 per cent of the cases. One may occasionally miss this sensation if the artery is pierced laterally or if there is some reaction around the artery from previous procedures or inflammatory conditions. The latter might prevent free movement of the artery in the soft tissue of the neck and thus obscure the snapping effect described.

If the snap cannot be felt, one has to resort to the blind penetration method. To determine whether the bevel of the needle is well placed in the artery and the angiographic procedure satisfactory, the following signs are helpful:

1. The patient experiences burning pain in the face and the back of the eye on the side of the injection when the contrast medium is introduced. The grimacing effect usually appears immediately after the injection has been completed and the maximal amount of pain is experienced during the late arterial phase and the capillary phase. When there is extravasation of contrast medium in the neck, the pain is experienced immediately during the injection and localized in the neck.
2. There is lack of swelling in the neck when saline solution is forcibly injected prior to the injection of the contrast medium.
3. A rapid return of the arterial blood into the syringe occurs immediately after the contrast medium has been injected.

Utilizing the above mentioned technique, we are now able to obtained satisfactory serial angiograms, using the Chamberlain bi-plane stereoscopic serial angiographic unit, in over 95 per cent of cases on the first attempt.

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ABDOMINAL VENOGRAPHY*

TECHNIQUE OF ROENTGEN VISUALIZATION OF THE INFERIOR VENA CAVA

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IN 1935 dos Santos[2] described a method of visualizing the inferior vena cava roentgenographically by injecting concentrated diodrast through the surgically exposed saphenous vein. Perhaps because of his failure to demonstrate a known lesion of the inferior vena cava by this method, the procedure received no particular recognition, and it was eleven years before abdominal venography was again reported.[7] In 1947 Fariñas[3] and O'Loughlin[6] independently described their techniques for roentgen visualization of the inferior vena cava. Fariñas cannulated the saphenous vein in the thigh after surgical exposure, while O'Loughlin, a radiologist, departed from surgical methods and performed percutaneous femoral vein puncture unilaterally. He used an 18 gauge needle and injected 20 cc. of 35 per cent diodrast at the rate of 1 cc. per second. He also described the use of sphygmomanometer cuffs on the thighs to prevent reflux of contrast medium down the legs. In addition, he had the patient employ the Valsalva effort during roentgen exposure in order to further fill the inferior vena cava. Fariñas employed an inflated balloon over the epigastrium to achieve the same result. In 1950 Stable[9] reported bilateral percutaneous femoral vein puncture with 16 gauge needles and the injection of 60 cc. of 50 per cent diodrast. Olivier[5] used the same method together with inflated cuffs on the thighs.

Although abdominal venography will probably never be widely adopted as a diagnostic method owing to its relatively rare indications, it may be of great value in demonstrating caval and pericaval pathology. Since complete or incomplete obstruction of the inferior vena cava commonly causes no clinical signs or symptoms, the diagnosis of such conditions is rarely made outside of the operating or autopsy rooms. It is this hiatus in diagnosis which may be filled by abdominal venography.

The indications for abdominal venography are as follows: To determine the presence of intrinsic obstruction of the inferior vena cava by blood clot or tumor thrombus; to determine the presence of partial or complete vena caval block by extrinsic pressure caused by abdominal and retroperitoneal tumors or by pericaval lymph node enlargements; to outline retroperitoneal tumors for surgical treatment or roentgen therapy; to outline caval and renal or ureteral anomalies. The greatest sphere of usefulness for abdominal venography will probably be in urologic diagnosis because of the frequency with which renal and adrenal tumors invade the renal and adrenal veins with extension of tumor thrombi into the vena cava, and because of the common involvement of the preaortic and pericaval lymph nodes by testicular tumors. A study of the usefulness of abdominal venography as an aid in urologic diagnosis has appeared elsewhere.[1]

METHODS

Three methods of visualizing the inferior vena cava roentgenographically have been employed and evaluated. The first method was adopted from those described by O'Loughlin, Stable, and Olivier, but has been modified in some respects. The patient is prepared by catharsis as for intravenous urography. Preliminary sedation with barbiturate and morphine or demerol is desirable. Both groins are shaved and prepared with skin antiseptics. Blood pres-

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sure cuffs are applied to the thighs above the knees and a preliminary scout film is taken of the abdomen. Using 1 per cent novocaine, skin wheals are raised over the femoral veins approximately one finger breadth below the inguinal ligaments and just medial to the pulsations of the femoral ateries. It is helpful to infiltrate the area deeply to minimize any pain which may occur with extravasation of contrast medium outside of the vein. The femoral veins are punctured with no. 16 gauge short bevel needles attached to syringes. When a free flow of blood is obtained hemostats are applied to the needles next to the skin to immobilize them. The needles are then connected to the limbs of an hypodermoclysis set which has been attached to a bottle of normal saline solution. It has been found that the addition of the infusion set is of considerable help. It provides a method of determining whether the needle is properly placed in the vein; it maintains the patency of the needle, since a slow infusion of saline is allowed between injections of the contrast medium; and it allows injection of the medium into the tubing rather than into the needle, thereby minimizing the chance of dislodging the needle during the injections. Using no. 18 gauge needles attached to 30 cc. syringes, 25 cc. of urokon 70 per cent is injected into each limb of the hypodermoclysis tubing 8 to 10 inches above the needles in the femoral veins. The tubing is clamped proximal to the site of injection. (Disposable plastic sets provided for hypodermoclysis have a segment of rubber at their distal ends through which the contrast medium is injected.) Prior to the injection of the contrast medium the cuffs are inflated approximately to diastolic blood pressure; the patient is asked to hold his breath, and to strain slightly against the closed glottis. Extremely rapid injection of the contrast material is not necessary, but it should be injected simultaneously on both sides in a period of fifteen to twenty seconds. Following completion of the injection the needle is withdrawn from the tubing and the infusion is allowed to flow into

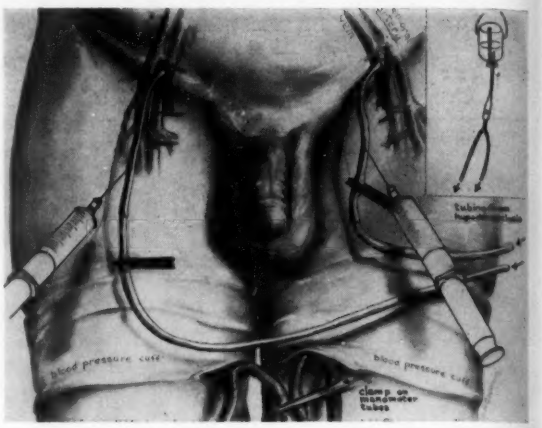

Fig. 1. Bilateral percutaneous femoral venipunture method of obtaining abdominal venograms.

the femoral veins. A roentgenogram at 1/5 second is made approximately five to eight seconds following the injection of the contrast medium. This allows the operator to leave the area of radiation hazard. This method is demonstrated in Figure 1. The use of the infusion allows reinjection for additional films if desired. It is not advocated

Fig. 2. Normal vena cavagram obtained by femoral venipuncture method. Early reflux into internal iliac and ascending lumbar veins is commonly seen with the Valsalva maneuver.

that the procedure be repeated more than once, since the total dose of urokon should probably not exceed 100 cc. There is usually a flush reaction due to rapid injection of contrast medium, but we have seen no vomiting, retching or laryngeal spasm in 50 cases performed by this method. Extravasation of the contrast material occurs in approximately 20 per cent of cases and is associated with local pain which can be effectively counteracted by the injection of 5 per cent to 10 per cent novocaine. The employment of the hypodermoclysis set as described above reduced the incidence of extravasation. Figure 2 shows a normal vena cavagram obtained by this method. The external and common iliac veins and the entire abdominal vena cava are well filled. There is slight reflux of medium into the internal iliac veins caused by the Valsalva effort. This amount of reflux is not considered abnormal. Figure 3 is a vena cavagram demonstrating a large tumor thrombus in the upper abdominal cava from a hypernephroma previously treated by right nephrectomy. More extensive reflux into the internal iliac veins and their branches is evidence of probable partial obstruction. Filling of the ascending iliolumbar-vertebral veins is more conclusive evidence of caval obstruction and collateral venous return.

Fig. 3. Vena cavagram (femoral venipuncture method) showing large filling defect caused by renal carcinoma extending into vena cava.

A second method of abdominal venography which we have employed in one case may be useful in demonstrating lesions of the upper abdominal vena cava. This method is a modification of that described by dos Santos[1] for translumbar aortography. The technique is modified by introducing the needle on the right side and into the vena cava rather than on the left side and into the aorta. Following adequate premedication, 1 per cent novocaine is infiltrated into the skin 3 to 4 inches below the twelfth rib and 4 finger breadths to the right of the midline. Deeper infiltration of novocaine is then done, down to the vertebral bodies. A no. 17 gauge 7-inch needle is advanced to a point just lateral to the vertebral column. A syringe attached to the needle is used to determine when the needle is in the vena cava. When venous blood is freely aspirated, the needle stop is advanced to the skin and set on the shaft of the needle. This prevents displacement of the needle during subsequent manipulation. A test injection of 2 to 3 cc. of urokon 70 per cent is helpful in determining the proper location of the needle. The solution can be injected through intravenous tubing since the rapid injection used for aortography is not necessary. Using blood pressure cuffs on the thighs and the Valsalva maneuver as previously described, injection of 25 cc. of urokon 70 per cent is done into the tubing and a roentgenogram is made one to two seconds after completion of the injection. The use of a rapid cassette changer is not necessary, since critical timing for the exposure is not required. This method is shown in Figure 4. Figure 5 demonstrates a normal vena cavagram obtained by the translumbar route. The infra- and supra-diaphragmatic segments of the vena cava are well demonstrated.

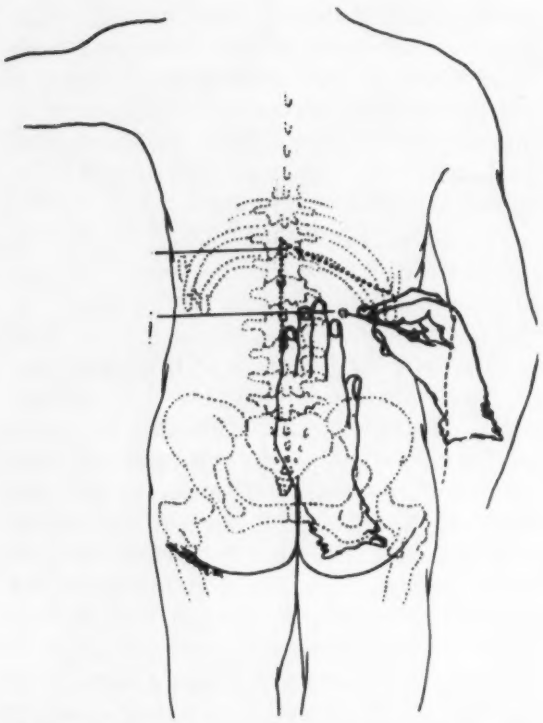

Fig. 4. Translumbar method of obtaining vena cavagrams. Needle should be introduced 3 to 4 inches lower than indicated in artist's drawing if visualization of the entire abdominal vena cava is desired.

A third method employed is a modification of the percutaneous femoral artery catheterization for aortography described by Pierce.[8] After adequate premedication and local infiltration anesthesia at the site of puncture just below the inguinal ligament, a no. 13 short beveled needle or a no. 12 gauge Robb cannula is introduced into the femoral vein just medial to the artery. When a free flow of venous blood is obtained, polyethylene tubing with an outside diameter of 0.075 mm. is then passed through the cannula for a distance of 10 to 12 cm. The cannula is withdrawn and pressure applied over the puncture site. The tubing, previously fitted with a blunt-end 16 gauge needle, is then attached to an intravenous infusion set. A test injection of 2 to 3 cc. of contrast medium followed by roentgen exposure will determine the correct location of the plastic tubing within the vein. If the plastic tubing does not thread easily it should not be forced. Blood pressure cuffs and the Valsalva maneuver are used as described earlier, and 30 cc. of contrast medium is injected into the intravenous tubing or directly into the 16 gauge needle attached to the polyethylene tubing. The latter is permissible since manipulation is safe when the tube has been threaded a short way up the iliac vein. Roentgenograms are made as described for the first method. This method allows movement of the patient and is particularly valuable when lateral or oblique projections are desired, and has the same advantages demonstrated by Walter and Goodwin[10] in use of transfemoral aortograms. Figure 6 is a vena cavagram obtained by this method with the patient in the right posterior oblique position. It shows a partial block of the inferior vena cava secondary to tumor thrombus originating from a hypernephroma of the left kidney. The pelvic collateral circula-

Fig. 5. Normal vena cavagram obtained by translumbar puncture method. Infra- and supradiaphragmatic segments of the vena cava are visualized.

Fig. 6. Vena cavagram obtained by femoral vein catheterization method with patient in oblique position. Pelvic collateral venous circulation is evidence of partial caval obstruction. Patient had renal tumor thrombus extending into vena cava above renal veins.

tion is well demonstrated and is evidence of the presence of caval obstruction.

The only definite contraindications to abdominal venography are: sensitivity to organic iodine; the presence of active thrombophlebitis in the leg or pelvic veins.

CONCLUSIONS

Bilateral percutaneous femoral vein puncture is the simplest method of visualizing the inferior vena cava roentgenographically. It can be carried out on the ambulatory patient. However, it does not allow movement of the patient without the risk of displacing the needle. The direct caval puncture method does not provide delineation of the lower vena cava and its anastomotic channels. Furthermore, it is probably more hazardous than the other methods since it is not possible to detect post-puncture bleeding from the vena cava and it is impossible to apply compression to prevent such extravasation.

Catheterization of the femoral vein through a needle or Robb cannula is somewhat difficult, but has the advantage that danger of perivenous extravasation is minimized and that movement of the patient in order to obtain additional projections is possible. It is felt that all three methods deserve further trial since considerable information can be obtained in many cases where their use is indicated. Such information is unobtainable by any other nonsurgical methods and may alter the treatment and prognosis of patients with caval and pericaval pathology.

SUMMARY

Abdominal venography, as described in 1935, has not yet obtained general acceptance or widespread use. This may be due, in part, to its infrequent indications but it may also be due to the technical difficulties generally attributed to the methods of outlining the vena cava roentgenographically. Three methods for the roentgen visualization of the inferior vena cava are described in detail: bilateral percutaneous femoral vein puncture, direct translumbar caval puncture, and percutaneous femoral vein catheterization. Each method has certain advantages, disadvantages, and indications. The percutaneous femoral venipuncture method is simple to perform and will give satisfactory results in the diagnosis of obstruction of the vena cava by blood clot, tumor thrombus or extrinsic compression. Information gained by abdominal venography is often unobtainable by other clinical methods. The findings may frequently alter the treatment and prognosis of patients with renal, adrenal and testes' tumors and may aid in the definition of other retroperitoneal lesions.

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A DEMONSTRATION BY RETROGRADE AORTOGRAPHY OF MULTIPLE PREGNANCIES IN THE DOG

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VISUALIZATION of the human placenta during late pregnancy, following aortography by direct puncture of the abdominal aorta through the lumbar route, was described by Coutts and co-workers[3] in 1935. Some of the placental sinuses and other vascular structures ascribed to the fetal circulation were shown. Employing a similar technique, Hartnett[7] analyzed 68 aortograms of pregnant women, and demonstrated 2 cases of placenta previa.

Identification of the pelvic arteries following injection of a contrast medium into the exposed femoral artery was made in 1932 by Saito and Kamikawa.[10] The retrograde femoral method for opacification of the abdominal aorta and its branches was explored by Fariñas[5] in 1941. Percutaneous retrograde femoral arteriography and aortography in obstetrics and gynecology were reported by Borell, *et al.* in 1952.[1] Placental sinuses were detected as early as the second month of pregnancy. The site of placental implantation was clearly visualized and the usefulness of this technique in the diagnosis of extra-uterine pregnancy and placenta previa was discussed. Whereas induction of labor has been mentioned following lumbar aortography,[7] which is usually done under general anesthesia, this complication has not been described after retrograde injection under local infiltration anesthesia.

Other methods studied in animals, but not applied to humans, include placentography performed by Ehrhardt and co-workers,[4] in the mouse, rabbit, and guinea pig, following the injection of contrast media directly into the heart or superior vena cava or by storage of thorotrast.

DEMONSTRATION

Retrograde femoral aortography has comprised a standard step in the investigation of aortic grafts and suture lines ever since the use of blood vessel transplants was introduced in human surgery by Gross, *et al.*[6] in 1947. The roentgenograms discussed in the present study were obtained during an analysis of the comparative effects of different suture materials on the growth of aortic anastomoses, con-

FIG. 1. Retrograde femoral aortogram in puppy. Metal clips mark aortic suture line.

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Aided by a Grant from the Playtex Park Research Institute.

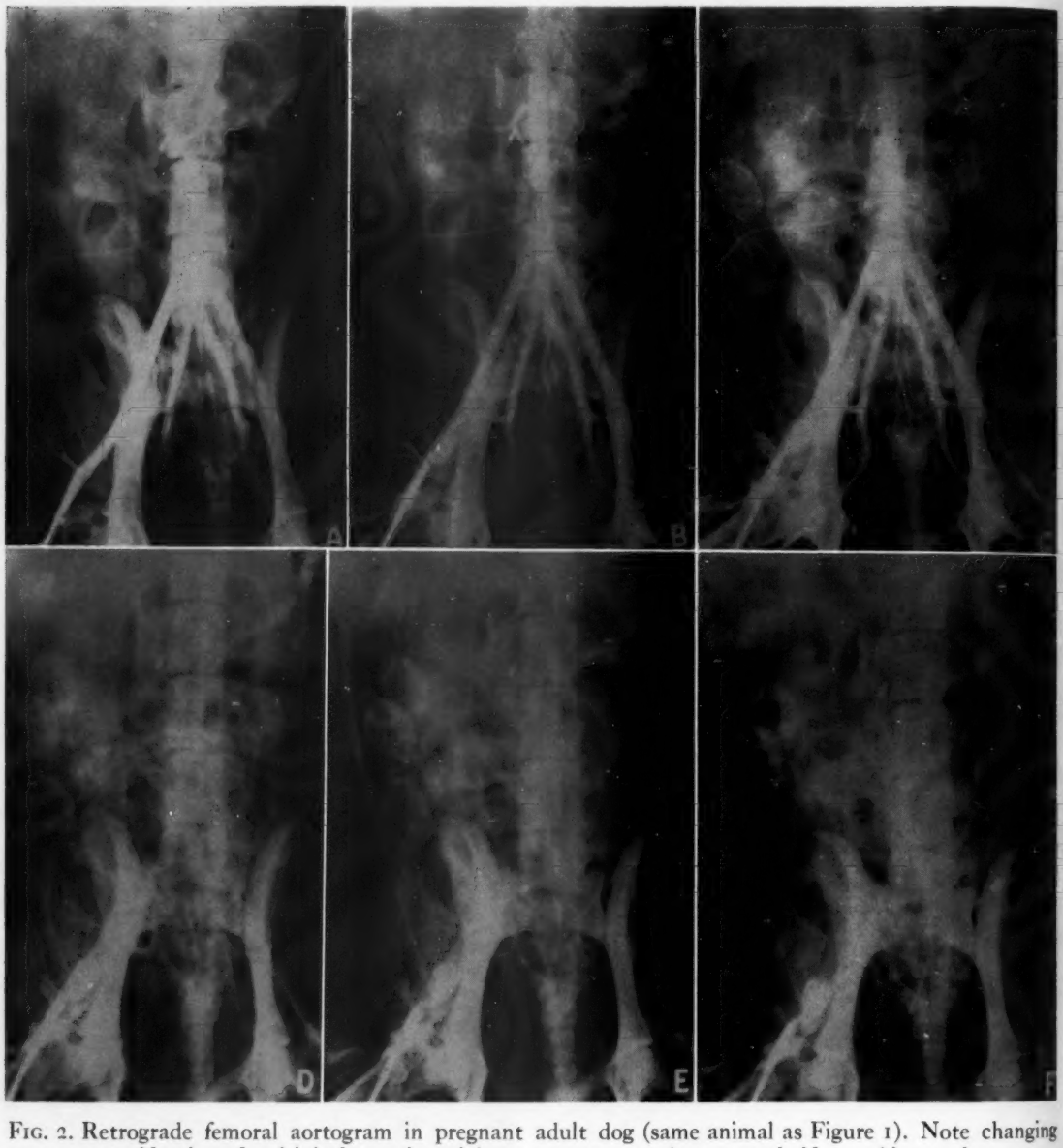

FIG. 2. Retrograde femoral aortogram in pregnant adult dog (same animal as Figure 1). Note changing opacification of multiple fetuses in serial roentgenograms taken at one-half second intervals.

structed in puppies that were sacrificed after reaching adult size.[8] The technique employed, as described in detail in an earlier publication,[9] consisted of injecting 30 cc. of 35 per cent diodrast rapidly in retrograde fashion into a cannulated femoral artery, and taking serial films at one-half second intervals with the Fairchild $9\frac{1}{2}$ by $9\frac{1}{2}$ inch magazine roll camera.

An initial aortogram was done 40 days after the lower abdominal aorta had been transected and the ends reunited with interrupted sutures of No. 00000 medium chromic catgut.* At that time the puppy weighed 5 pounds; no unusual vascularization of the genital circulation was observed (Fig. 1).

Aortography was repeated 223 days later, at which time the full grown dog

* The suture material was supplied through the generosity of the Ethicon Suture Laboratories, Inc., New Brunswick, New Jersey.

weighed 25 pounds. Increase in diameter at the anastomosis was commensurate with the growth of the remainder of the aorta (Fig. 2*A*). Unexpected opacification of the genital circulation was encountered, with the demonstration of multiple fetograms (Fig. 2,*B–F*). While one could not separately identify the placental and fetal vessels for each fetus, it was noted that there was an ascending and descending intensity of opacification of each fetus on the serialograms. This is highly reminiscent of the "staining" seen in nephrograms or in highly vascular brain tumors visualized by means of cerebral angiography.

Rapidly exposed serialograms covering the arterial, capillary, and venous phases of the circulation of the pregnant uterus may permit recognition of a vascular "stain," which could be of value in the early diagnosis of multiple pregnancies before the fetal skeleton is visualized or the fetal heart sounds are noted.

In addition to this demonstration of multiple canine pregnancies, retrograde aortography has contributed to the study of placenta previa, extra-uterine pregnancy, and the differentiation of intra- from extra-uterine pelvic masses.[1] An additional significant procedure in the investigation of these problems may be the identification of the placental site by a radioactive isotope technique as described by Browne, *et al.*[2]

SUMMARY

1. Retrograde femoral aortography has been demonstrated to be of value in the study of the placental and fetal circulations in humans and in the experimental animal.

2. An example of multiple pregnancies in a dog is presented.

3. A "staining" type of opacification of each fetus is noted, similar to that observed in nephrograms and in cerebral angiograms of highly vascularized brain tumors.

4. This method offers a means for the very early recognition of multiple pregnancies.

APPENDUM

Since this article was submitted for publication, additional material on the subject has appeared in the article entitled "Pelvic Arteriography As a Diagnostic Procedure in Early Intrauterine Pregnancy and Tubal Pregnancy" by U. Borell, I. Fernström and A. Westman in *Geburtsh. u. Frauenh.*, 1955, *15*, 11.

The authors gratefully acknowledge the technical assistance of Mr. Ruthven C. Ferreira and Mr. Edmund Schmidt.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Published by: CHARLES C THOMAS, PUBLISHER, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued monthly. Annual subscription: United States, Mexico, Cuba, Central and South America, $15.00; Canada, $16.00; other countries, $17.00. Current single numbers, $1.50. Advertising rates submitted on application. Editorial office, 110 Professional Building, Detroit 1, Michigan. Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois. Information of interest to authors and readers will be found on page vi.

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EDITORIAL

NEW HORIZONS IN CARDIOVASCULAR ROENTGENOLOGY

CURRENT, essentially technical efforts to expand the usefulness of angiography include the following approaches:

1) *Serial Short Exposure Techniques.* Blood in the heart and arteries moves as fast as 100 cm. per second. Roentgenographic exposures of 1/60 second are not short enough to "stop" the movement of intra-arterial or intraventricular contrast agents or rapidly moving anatomic structures such as valves. By radical changes in timing equipment for roentgenography, exposures as short as one millisecond (1/1,000 second) duration have become a practical reality. By minimizing blurring due to movement, such short exposures improve roentgenographic quality and increase the diagnostic yield while at the same time they allow a reduction in the radiation hazard and the dose of contrast agent needed. Serial short exposure studies represent an effort to minimize or at least control the effect of motion.

(2) *Cineroentgenography.* Here the emphasis is on the study of motion itself. Image intensification techniques have already greatly facilitated cardiovascular studies. By means of very high exposure frequencies (in the order of 500–1,000 frames per second) "slow motion" cineroentgenography may reveal phenomena hitherto invisible due to motion (as did Prinzmetal's high speed photographic movies of the beating dog heart). Technical achievements already are sufficient for ultra high speed cineroentgenography in small experimental animals while further progress should allow application to human subjects.

(3) *Image Magnification.* When adequate image intensification systems have replaced the presently available crude prototypical devices, it should be possible to employ magnification techniques to static and cineroentgenographic contrast cardiovascular studies. If in this manner the radiologist is allowed to study the form and function of man's capillaries, it is certain that important contributions to research and diagnosis will result. The *in vivo* roentgenhistologic diagnosis of tumors may become an exact and reliable procedure. Many obstacles remain, but from a theoretical point of view the energy requirements for image magnification are not insurmountable.

(4) *"Particulate" Contrast Cardiovascular Agents.* The development of a safe agent affording punctate zones of contrast rather than the familiar bolus of radiopaque fluid represents a promising area for research. Appropriate roentgenographic studies of discrete, tiny dots of contrast medium within the cardiovascular system will afford an exact assessment of intracardiac and intravascular blood-flow and turbulence (similar to snowflakes in a headlight beam). When it becomes roentgenologically possible to visualize turbulence, the stethoscope may indeed be administered a "mortal blow." Since heart murmurs are but an audible manifestation of turbulence, it will become possible for the radiologist to find, quantitate, and demonstrate the exact source of murmurs. In addition, punctate ("particulate") radiopaque visualization offers a method for measuring blood velocity and flow. The cardiac output can therefore be determined as a function of cardiac contraction, and observations can be repeated at rapid intervals. The percentage of blood-flow entering various branches of the pulmonary artery can thus be estimated by simple particle counts. Furthermore, the absolute flow in vessels of measurable caliber could be calculated from averaged measurements of particle-travel occurring during short exposures of known length (an expression of blood velocity).

While the technique is not yet a practical reality, the above considerations appear to rest upon a fundamentally sound basis. It is justifiable on the basis of current investigations to predict the eventual availability of one or more agents capable of producing punctate radiopacification with safety. Agents under active study at this time include solid and semisolid particles (the short lives of which appear to prevent damage due to pulmonary embolization), droplets of fluid, or even bubbles of gas carried within a bolus of radiopaque fluid. Droplets of radiopaque fluid which retain their shape and sharply defined borders during passage through the right heart and pulmonary arteries, and yet which do not produce measurable pulmonary embolization, offer the best opportunity. Maximum realization of the potentialities of such agents will involve special techniques for roentgenographic exposures. The customary 1/60 second study represents the photographic sum of two exposures, each approximately one-quarter cycle in duration and separated by a silent interval of about one-half cycle. If, in the study of blood-borne radiopaque particles, two 0.5 millisecond roentgen-ray pulses, separated from each other by an interval of one millisecond, are employed, measurement of the distance between corresponding parts of the resultant roentgenographic double image will provide a reasonably precise indication of the average particle-velocity during the brief (2 millisecond) period of observation. Through serial studies, velocity can be plotted as a function of phenomena such as the electrocardiogram and blood pressure. Blood flow (volume per unit time) can be similarly derived. The use of more than two impulses will allow the calculation of acceleration. It is hoped this technique will soon be applied to man with safety.

The potentialities of cardiovascular roentgenology warrant continued investigative effort by academic and industrial scientists, engineers and chemists. Roentgenology has the opportunity to make really important contributions to cardiovascular medicine. The necessary enthusiasm already exists within our ranks. If encouragement and financial support are forthcoming, the objectives are by no means nebulous.

Charles T. Dotter, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS*

JAMES PICKER FOUNDATION PROVIDES FUNDS FOR RESEARCH

On behalf of the James Picker Foundation, the National Academy of Sciences-National Research Council announces the continued availability of funds in support of radiological research.

Applications for the fiscal year 1957–1958 must be submitted on or before December 1, 1956. Further details and application blanks may be obtained from the Division of Medical Sciences, Room 310, National Academy of Sciences-National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.

Grants for Scholars of $6,000 per year will be made directly to the Scholar's institution as a contribution to his support, or his research, or both. Initial grants are limited to one year, but renewal may be recommended. Applications should be submitted by the institution on behalf of the candidate.

Fellowships in Radiological Research are open to candidates seeking to gain research skills leading to investigative careers in the field of radiology. While persons from closely related disciplines are eligible to apply, candidates whose training has been directly in the field of radiology will receive preference under this program. Candidates must hold the M.D., Ph.D., or Sc.D. degree or the equivalent, and should ordinarily be not more than thirty-five years of age.

UNIVERSITY OF MINNESOTA OFFERS RADIATION THERAPY COURSE

The University of Minnesota announces a continuation course in Radiation Therapy for Radiologists which will be held at the Center for Continuation Study and the adjacent Museum of Natural History Auditorium on the University campus from November 5 to 9, 1956. Radiation physics and biology will be considered, and two major groups of neoplastic diseases will be covered in detail, gynecological tumors and tumors of the maxillofacial area, pharynx and larynx.

Guest speakers will be Doctors Hans-Ludwig Kottmeier, Director, Gynecological Department of Radiumhemmet, Stockholm, Sweden; J. A. del Regato, Director, Penrose Cancer Hospital, Colorado Springs, Colorado; Henry Kaplan, Professor of Radiology, Stanford University School of Medicine, San Francisco, California; John B. Graham, Instructor in Gynecology, Harvard Medical School, Boston; Titus C. Evans, Radiation Research Laboratory, State University of Iowa College of Medicine, Iowa City; and Harold Tivey, Jefferson Davis Hospital, Houston, Texas. Dr. Kottmeier will also present the annual Leo G. Rigler Lecture on Wednesday evening, November 7. The course will be presented under the direction of Doctors Leo G. Rigler, Professor and Head, Department of Radiology, and Donn G. Mosser, Assistant Professor and Director, Division of Radiation Therapy.

AMERICAN BOARD OF RADIOLOGY ANNOUNCES EXAMINATIONS

The Spring, 1957 examination will be held at the Hotel Floridan, Tampa, Florida, April 1–6, inclusive. The deadline for filing applications for this examination is January 1, 1957. The Fall, 1957 examination is scheduled for the Shoreham Hotel, Washington, D. C., September 23–28, inclusive. The deadline for filing applications for this examination is June 1, 1957.

Applications should be addressed to Dr. B. R. Kirklin, Kahler Hotel Building, Rochester, Minnesota.

* The list of Meetings of Radiological Societies appears in the September, 1956, issue of the JOURNAL, and will be published in the November number.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

THE AUXILIARY HEART. By William Walter Wasson, M.D. Cloth. Price, $10.50. Pp. 184, with 72 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1954.

Dr. Wasson has drawn on twenty-five years' experience and an extensive review of the literature to evaluate the importance of respiration on the propulsion of blood into and out of the lungs. He details the microscopic anatomy of the air sacs along with their accompanying capillaries and supporting structures, pointing out the importance of the elastic fibers in transmitting the negative and positive pressures to the lumina of the air sacs and their capillaries.

The physiology of respiration is summarized in as far as it affects the pressure in the lesser circulation and its contribution to propelling the blood into and out of the lungs. He stresses that the wall of the right ventricle increases very little in thickness after birth, while that of the left ventricle increases considerably. He concludes that although the left ventricle can propel blood through the greater circulation without outside help, the right ventricle probably cannot do the same with the lesser circulation. The blood in the lung capillaries and veins is propelled by the squeezing action of the lungs on the capillaries during expiration and suction during inspiration in a manner quite similar to that which propels lymph and blood in the systemic veins.

Many representative roentgenograms are presented showing the many chest diseases which may influence the lesser circulation. He divides them into four groups: I. Diseases of the heart; II. Diseases of the pulmonary arteries, capillaries and veins; III. Diseases of the lungs involving the lesser circulation; and IV. Diseases of the thorax and general state of the patient.

The effect of diaphragmatic motion on the lesser circulation is discussed. The importance of relatively normal diaphragmatic motion to handling of chronic pulmonary congestion is discussed and cases are presented.

An extensive historical sketch of the discoveries and theories about the lesser circulation is given, along with the author's personal comments on many of the discoveries. The book should be of as much, if not more, interest to internists and surgeons as to radiologists.

FRANK M. WINDROW, M.D.

STUDIES IN HYPERTENSIVE CARDIOVASCULAR DISEASE WITH SPECIAL REFERENCE TO CARDIAC FUNCTION. The Scandinavian Journal of Clinical and Laboratory Investigation, Supplement 17. By Edvardas Varnauskas. Pp. 116, with 11 illustrations and 14 tables. The Scandinavian Journal of Clinical & Laboratory Investigation, Medical Department A., Rikshospitalet, Oslo, Norway, 1955.

The literature is reviewed and the physiologic laboratory experience is presented on 84 patients and 9 normal subjects. Essential hypertension was classified according to the criteria of the New York Heart Association. Heart volume was measured by roentgenograms with the patient standing. The mean value was 450 ml./m.^{2} in groups I and II and 680 ml./m.^{2} in groups III and IV. Electrocardiograms were classed as normal left axis, abnormal T or ST, fibrillation and conduction defects. No known cases of myocardial infarction were included. The more severe changes occurred in groups III and IV. The mean cardiac index l/min./m.^{2} was lowest in groups III and IV. The mean stroke index, ml./m.^{2} was also lowest in groups III and IV and lower than normal. This is associated with the higher mean pulse rate in groups III and IV. No parallel decrease in stroke index was noted with progress in the eye ground changes.

The mean pulmonary artery pressures were 11, 19 and 38 respectively for normal, I and II and III and IV groups. The pulmonary artery pressure was found to correlate with a rise in heart volume. Enlargement beyond 600 ml./m.^{2} was usually combined with pathologically elevated pulmonary artery pressure.

The average mean pulmonary capillary venous pressures were 6, 6 and 11–22, respectively, for normal, I and II and III and IV group

experience. The correlation revealed that pulmonary capillary venous pressure exceeding 12 mm. Hg was not recorded in any cases with heart volume equal or less than 490 ml./m.^{2} The presystolic and systolic waves were usually marked in cases of hypertension and sometimes were of the order found with mitral stenosis, although this lesion was absent in 3 cases at autopsy.

The brachial arterial pressures were 118, 77, and 94 for average systolic, average diastolic, and the mean respectively. The average of all mean pressures was about 150 in all hypertensive groups with a range of 90 to 232. On the average systolic, diastolic and mean pressure were the highest in cases with heart volume between 450 to 600 ml./m.^{2}

The right atrial pressure exceeded 5 mm. Hg in all cases but one in which the heart volume was greater than 600 ml./m.^{2} The pulmonary vascular resistance in dynes sec. cm.$^{-5}$ showed considerably higher mean values (200) in cases with a heart volume exceeding 600 ml./m.^{2} than in others in which the spread and mean values (51) approached normal limits.

The peripheral vascular resistance in dynes sec. cm.$^{-5}$ showed the normal mean of 909, groups I and II about 2,000, and groups III and IV about 2,600. The left ventricular work index at rest in kg.m./min./m.^{2} showed a mean of 6.2 for normals about 8.0 for groups I and II, about 6.2 for groups III and IV. A correlation of the heart volume against the left ventricular work, showed a progressive decrease in work with increasing heart size. On the other hand, right ventricular work correlation showed a progressive increase in work against increasing heart size up to 700 ml./m.^{2} and then it decreased.

Para-aminohippurate clearances in ml./min./m.^{2} showed a mean of 354 for normals, 222 for groups I to II and 118 for groups III to IV. The differences appear significant. The inulin clearance, ml./min./m.^{2} showed a mean of 75 for normals, 55 for I and II and 41 to groups III and IV. The filtration fraction in per cent showed a mean of 21 for normal, 24 for I and II and 34 for III and IV hypertensives.

Exercise, lanatoside C_1 and hexamethonium bromide studies were also evaluated. The validity of the given exercise tests is discussed. Lanatoside C_1 showed an increase of total blood flow and total ventricular work associated with a decrease in filling pressure index for both ventricles. Hexamethonium caused a lowering of the peripheral resistance, and total left ventricular work index in 3 cases without left ventricular failure and an increase in 1 case with left ventricular failure.

Jan Nyboer, M.D.

Diagnosis of Congenital Heart Disease. By Sven R. Kjellberg, Edgar Mannheimer, Ulf Rudhe, and Bengt Jonsson. Cloth. Price, $22.00. Pp. 649, with 581 illustrations. Year Book Publishers, Inc., 200 E. Illinois Street, Chicago 11, Illinois, 1955.

The cardiologic team of the Pediatric Clinic of the Caroline Hospital of Sweden in this volume presents the results of its indefatigable efforts in the diagnosis of congenital heart disease. The book is organized along standard lines, with discussion of cardiac embryology, roentgenologic anatomy and physiology of the heart, as well as special diagnostic techniques such as phonocardiography, electrocardiography, electrokymography, cardiac catheterization, and angiocardiography.

The text also includes a section on the various congenital cardiac defects, both cyanotic and acyanotic in type. These are illustrated by actual cases and in most instances the roentgenologic, particularly the angiocardiographic, aspects are in prominence. The angiocardiograms, of course, are most noteworthy. The team employs selective angiocardiography almost exclusively and their demonstration of the anatomic features of congenital heart disease in the living patient is unparalleled in medical literature. Their illustrations of the angiocardiograms correlated with postmortem findings in the same patients can add greatly to the average radiologist's understanding of the congenital heart defect under discussion. This beautiful exhibition of the potentiality of contrast studies in the diagnosis of congenital intracardiac defects should dispel all doubts as to the value of angiocardiography in this respect.

If any criticism can be leveled at this monumental work, it is the surprising manner with which the vascular rings are discussed. Only one and one-half pages of the text are assigned to this important type of anomaly, and the only illustration is that of a retro-esophageal right aortic arch. Surely more attention should be paid, at least from the standpoint of the radiologist, to these vascular anomalies which so commonly accompany intracardiac defects.

The entire volume is well written in good

English and the bibliography is international in scope. Certainly everyone interested in the diagnosis and treatment of congenital heart disease should own this book.

Harold Fulton, M.D.

Die Röntgen-Untersuchung des Herzens und der Grossen Gefässe. Von Prof. Dr. R. Janker, Prof. Dr. Grosse-Brockhoff, Prof. Dr. R. Haubrich, Dr. H. Lotzkes, Dr. A. Schaede, und Dr. H. Hallerbach. Cloth. Price, DM 34.50. Pp. 222, with 249 illustrations. Verlag W. Girardet, Neumarkstr. 50, Wuppertal-Elberfeld, Germany, 1955.

This book consists of a group of lectures given during a brief roentgenology course at Bonn, Germany, in 1954 with each chapter written by the lecturer. The first chapter on technique of roentgenologic study of the heart discusses the different methods of examination, beginning with fluoroscopy and orthodiagraphy, then leading to spot films and laminagraphy. Special consideration is given to transverse laminagraphy, a method used particularly in Europe during the last few years. The different apparatus for serial roentgenography and cineroentgenography are described and modern equipment for rapid angiography done simultaneously in two planes is illustrated. At the end of the chapter there are useful tables relating to the roentgen ray exposure and skin dose received by the patient during the different types of examinations.

The second chapter, entitled "Electrocardiogram and X-ray Examinations," compares results of routine roentgenologic examinations of various heart lesions with those of clinical and electrocardiographic studies. The author has tried to evaluate the differential diagnostic indications and the hemodynamics as related to the heart. Particular emphasis is placed on pericardial effusions. Primary myocardial disease and cardiac infarcts are described, and the last part of the chapter deals with damage to the heart due to an overburdened circulation, such as in arteriovenous fistulas, congenital heart disease and hypertension. Most of the entities are illustrated by electrocardiograms and roentgenograms as well.

The third chapter on "Electrokymography" shows, with drawings and tracings, the usefulness of this method. The technique is given and its application in several examples, such as myocardial disease, old infarcts and pericardial disease, is described with cases and illustrations.

Chapter IV describes very briefly the technique of catheterization of the heart and of angiography. A section on the indications and results of such examinations includes a group of case descriptions with good illustrations of serial roentgenograms. Results and indications of cardiac catheterization, angiocardiography and retrograde thoracic aortography in congenital and acquired heart disease are discussed and mention is made of the applicability of these different methods. A number of roentgenograms depict the lesions in this chapter.

Another chapter encompasses a group of actual cases and their examination. The first step always is fluoroscopy. Additional methods are coordinated with the clinical aspect of the case. Catheterization, angiocardiography and thoracic aortography as a rule, should be carried out only if surgery is contemplated and should not be used for diagnostic purposes alone. The author considers it necessary, therefore, to use all conventional methods before resorting to these more formidable procedures.

The last chapter in the book is on cineroentgenography. In it the author refers to a book written by himself on this subject.

This volume is a useful introduction to roentgenologic examination of the heart and great vessels and, as such, is of value to the physician familiar with the German language. It is not to be considered a formal textbook.

Egon G. Wissing, M.D.

Cardiac Diagnosis; A Physiologic Approach. By Robert F. Rushmer, M.D., Associate Professor of Physiology and Biophysics, University of Washington Medical School. Cloth. Price, $11.50. Pp. 447, with 249 illustrations and 11 tables. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pennsylvania, 1955.

The role of pressure, sound, flow, electric and roentgen observations of the cardiovascular system has been excellently illustrated as a basis for a modern approach to dynamic as well as clinical diagnosis of the central cardiac status The exclusion of pertinent tables defining the criteria for normality and range of direct blood pressure measurements, blood flow, roentgen and other physiologic measurements definitely limits the usefulness of this didactic volume. Readers expect more than an empirical primer statement that "wide experience is the most important factor in this type of evaluation." Lord Kelvin once said, "I often say that

when you can measure what you are speaking about, and express it in numbers, you know something about it; but when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind; it may be the beginning of knowledge, but you have scarcely, in your thoughts, advanced to the stage of Science, whatever the matter may be."

JAN NYBOER, M.D.

HÄMODYNAMIK DES HERZENS IM RÖNTGENBILD, MIT BESONDERER BERÜCKSICHTIGUNG DER HERZKATHETERISIERUNG UND DER ANGIOKARDIOGRAPHIE. Von Priv.-Doz. Dr. P. Thurn, Bonn. Cloth. Price, DM 48.—. Pp. 167, with 81 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

This book is written primarily for the student of roentgenology. The hemodynamic information is limited to numerical pressures at the tip of the catheter and systemic blood flow data recorded from diverse clinical and surgical heart cases.

Electrocardiographic and blood pressure graphs are not shown or discussed. Numerous photographs of the catheter in situ for pressure recording are shown. A few analyses of oxygen content of the samples at various locations appear to be given for additional guidance to the diagnosis of congenital cardiac defects. Numerous angiocardiograms and roentgenkymograms illustrate the book.

The author emphasizes that changes in hemodynamics influence the mass and the specific blood volume of each heart chamber or blood vessel. Among the pathophysiologic factors which produce a change in roentgenograms are cardiovascular myogenic tonicity, pulmonary and systemic resistance to blood flow, the residual blood volume of each chamber in systole, the cardiovascular blood pressures at various sites, the functional status of each cardiac valve, the degree of locations and directions of the blood shunts, if present, and the regulative dynamic function of the associated chambers before and after increased work load is applied by a given heart case.

JAN NYBOER, M.D.

ELECTROKYMOGRAPHY; WITH SPECIAL REFERENCE TO VALVULAR, PULMONARY AND INFUNDIBULAR STENOSIS. Acta radiologica, Supplement 134. By Ulf Rudhe. Price, Sw. Kr. 30:-. Pp. 205, with 108 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

This monograph is a classic for its description of dynamics and events associated with the electrokymograms of the pulmonary artery pulsations. These studies were made on normals and 36 patients with infundibular stenosis. Abnormal configurations were observed in all such abnormal groups and differed from those with pulmonary stenosis. These deviations are produced by turbulence within the artery. The presence of a septal defect or overriding of the aorta does not modify the characteristic kymogram. No information could be obtained by this method regarding the degree of the stenosis nor the presence of other malformations.

JAN NYBOER, M.D.

PERIPHERAL VASCULAR DISEASES. By Edgar V. Allen, B.S., M.A., M.D., M.S. in Medicine, F.A.C.P., Section of Medicine, Mayo Clinic; Nelson W. Barker, B.A., M.D., M.S. in Medicine, F.A.C.P., Section of Medicine, Mayo Clinic; and Edgar A. Hines, Jr., B.S., M.A., M.D., M.S. in Medicine, Section of Medicine, Mayo Clinic; with Associates in the Mayo Clinic and Mayo Foundation. Second edition. Cloth. Price, $13.00. Pp. 825, with 316 illustrations. W. B. Saunders Company, West Washington Square, Philadelphia 5, Pennsylvania, 1955.

The second edition of this book should be in the library of every surgeon and every internist. First of all, peripheral vascular disease is an extremely important subject in which interest is constantly growing. Then there is a greater demand for information in this area, in view of the increased longevity of the people of the United States and other lands as well.

Excellently printed and bound, the volume contains a wealth of material such as can be found in no other single volume. There is a logical arrangement of the diseases of the vascular system, which makes this a useful reference book and one which is also easy to read.

The first three chapters are of particular interest and contain much basic information. The admonitions and precautions which are listed can well be heeded by anyone who is interested in this subject. The authors approach the subject in a practical way, condensing a vast amount of research and clinical work, with

distinct emphasis on the clinical nature of the various diseases discussed. Many excellent color plates, as well as black-and-white photographs, demonstrate the important features of each condition. At the end of each chapter is a helpful bibliography which the reader can use for further study and which gives credit to sources that have been used. There is also a short biography of outstanding medical men at the beginning of each subject, together with a photograph of the man himself and a short resume of his interest in the particular field. These biographies bring to the young physician a bit of medical history which adds to the interest of the subject and to the general information of the reader.

The reviewer has found the first edition of this book of great help in the study and in practice of a peripheral vascular nature. He knows of no other single volume which gives so much carefully condensed, accurate information. The second edition surpasses the first in many respects, both as to format and amount of information. It includes an up-to-date review of the vast advances that have been made in vascular surgery in the last ten to fifteen years. There is a complete discussion of the treatment of varicosities, aortic aneurysms, the use of grafts, and the care of patients requiring this type of surgery.

In the reviewer's candid opinion, this book is a must in every medical library. Experience has proven it a welcome gift to residents and interns and others interested in this field. The three authors are to be congratulated and thanked for their contributions.

Eugene A. Osius, M.D.

Klinische Elektrokardiographie. Von Dr. M. Holzmann, Zürich. Third edition. Cloth. Price, DM 80.—. Pp. 688, with 303 illustrations and 9 tables. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1955.

The author has succeeded in bringing up to date most of the accepted and forensic concepts of direct, semidirect and conventional electrocardiography. Vector analysis of the QRS and T components was treated cautiously. The clinical illustrations were often made with three simultaneous leads from different areas, but these were not controlled against a common reference lead. The contributions, merits and limitations of exploratory esophageal and intracardiac leads are not well defined. The electrocardiograms associated with valvular and congenital deviations of the heart, cardiac trauma and tumors, and systemic neuromuscular disorders, such as Friedreich's syndrome, are outlined but are not well illustrated. Extensive discussion and illustrations are given in regard to T-U deviations. The nomenclature used follows European characteristics. An atrial electrocardiogram is designated Eag and a ventricular electrocardiogram is designated Evg. Excellent tables and diagrams aid in defining the subject matter. Physicians familiar with German will find this clinical volume helpful.

Jan Nyboer, M.D.

Pathologic Physiology; Mechanisms of Disease. Edited by William A. Sodeman, M.D., F.A.C.P., Professor of Medicine and Chairman of the Department of Medicine, School of Medicine, University of Missouri, Columbia, Missouri. Second edition. Cloth. Price, $13.00. Pp. 963, with 173 illustrations. W. B. Saunders Company, 218 Washington Square, Philadelphia 5, Pennsylvania, 1956.

An attempt to show the relation between the fundamental physiologic and biochemical disturbances that occur in disease and the clinical picture resulting from these disturbances is a praiseworthy venture. The contributors to the second edition of this volume have, on the whole, been successful in avoiding long descriptions of normal physiology or clinical details in order to devote more space to the intermediate material which is the prime concern of the book.

As a large part of medical research, especially in recent years, has been directed toward understanding the very problems with which this book deals, it would be clearly impossible for the authors to include all the important findings. It is therefore inevitable that there will be some disagreement on their selection of material. For example, in the chapters on the cardiovascular system, the interesting circulatory changes caused by pregnancy and those produced by arteriovenous fistulas are perhaps discussed too briefly. In the section on endocrinology, more attention might have been given to recent advances in knowledge of the thyroid hormone, particularly in regard to tri-iodothipronine. The recent work of Christie and his collaborators on the efficiency of breathing in certain abnormal states probably merits fur-

ther discussion in the chapters on the lung.

In general, the book, including the aforementioned sections, is well written. The authors' efforts to keep the size of the book within reasonable limits are certainly not to be criticized. The style is generally clear. As this book deals with rather difficult subjects, its lucid style is no small advantage. The chapter on electrocardiography is a good example of straightforward writing that is far from dull. Readers wishing to gain further understanding of the mechanism of disease will undoubtedly find a study of this book rewarding.

Edward A. Carr, Jr., M.D.

Radiology for Medical Students. By Fred Jenner Hodges, M.D., Professor and Chairman, Department of Radiology, University of Michigan; Isadore Lampe, M.D., Professor, Department of Radiology, University of Michigan; and John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan. Second edition. Cloth. Price, $8.00. Pp. 439, with 105 illustrations. Year Book Publishers, Inc., 200 E. Illinois Street, Chicago 11, Illinois, 1954.

This work is evidently based on a large experience in teaching medical students and the text has been written with enthusiasm for the task. It consists of two parts, one dealing with diagnostic radiology and one with therapeutic radiology. The introduction briefly traces the development of radiology and the first chapter in Part 1 presents a highly satisfactory description of general radiology. Five chapters are devoted to the various anatomic divisions that may be subject to examination. Explanation of positions and interpretation are easily understood in most instances. Such complex procedures as angiography are appropriately succinct. However, some examinations of practical interest to the medical student have been slighted. The plain film of the abdomen, so often an early recourse in the investigation of acute abdominal symptoms, is rather neglected. Appropriate space has been allotted to diseases of the lung and pleura, but half of this has been devoted to tuberculosis, resulting in thin coverage of some common pulmonary problems that confront the student.

The discussion of the physical and biologic basis for radiotherapy is excellent. Description of the time-dose relationship, so essential to the rudiments of therapeutic radiology, is clear and uncomplicated. Seven chapters are devoted to malignant and nonmalignant diseases that may be treated by radiation. The clinical aspects are presented in a logical and credible manner, but the emphasis is on pathology of cancer rather than on the principles of radiotherapy and treatment planning.

This text is profitable reading for the medical student and will enable him to utilize radiologic services to good advantage in his own studies and practical clinical problems.

Vincent P. Collins, M.D.

Annual Review of Medicine. Volume 6. 1955. David A. Rytand, Editor. Cloth. Price, $7.00. Pp. 459, with tables. Annual Reviews, Inc., Stanford, California, 1955.

This series of reviews of recent advances in knowledge in selected fields of medicine is introduced by a pertinent and timely discussion of the problem of keeping up with medical literature. The individual chapters illustrate well the problem of the reviewer faced with the current plethora of medical writing. The individual reviews do not represent a mere catalogue of titles, but indicate careful selection of the useful and important contributions, with a synthesis of the important advances in such a fashion that the story catches the reader's interest.

Of particular interest to roentgenologists is the chapter on "Contrast Methods in Diagnostic Roentgenology," by B. Felson. Other subjects relevant to problems which the roentgenologist encounters daily in his diagnostic and therapeutic activities include: Diseases of the Cardiovascular System (Surgery); The Leukocytes and Leukopathies; Neoplastic Disease (Cancer); Diseases of the Bones and Joints; and Diseases of the Urinary System (Surgical).

The value of the reviews is enhanced by a bibliography for each chapter. In addition, an annotated list of reviews provides a valuable means of access to recent summaries on many medical subjects.

William D. Robinson, M.D.

BOOKS RECEIVED

Über die Technik der simultanen Telefilmplanigraphie. Acta radiologica, Supplement 137. By Vidar Backlund. Paper. Price, Sw. Kr. 25:-. Pp. 124, with 39 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

THE TREATMENT OF FRACTURES. Volume I. By Lorenz Böhler, M.D., Director of the Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Translated from the Thirteenth German Edition by Hans Tretter and others. Cloth. Price, $24.50. Pp. 1,104, with 1,721 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1956.

OBSERVATIONS ON THE MIGRATION OF SOME LABELLED SUBSTANCES BETWEEN THE URINARY BLADDER AND THE BLOOD IN THE RABBIT. Acta radiologica, Supplement 135. By Sven Erik Englund. Paper. Price, Sw. Kr. 25:-. Pp. 80, with 50 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

TOMOGRAPHIC STUDIES ON THE NORMAL AND INJURED KNEE. Acta radiologica, Supplement 138. By Stig Fagerberg. Paper. Price, Sw. Kr. 30:-. Pp. 93, with 51 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

NORME PER LE PROTEZIONI CONTRO LE RADIAZIONI IONIZZANTI. By F. Fossati, P. Gallone, L. Parmeggiani, C. Polvani, M. Scolari. Cloth. Pp. 259, with figures and tables. Ulrico Hoepli, Milan, Italy, 1956.

DIAGNOSTIC ROENTGENOLOGY. Ross Golden, M.D., Editor, Visiting Professor of Radiology, University of California at Los Angeles, Emeritus Professor of Radiology, College of Physicians and Surgeons, Columbia University; Formerly Director of the Radiological Service, Presbyterian Hospital, New York. Looseleaf. Renewal pages. Volume I. Chapter IV. Radiology of the Heart and Great Vessels. By Robert N. Cooley and Robert D. Sloan. Price, $15.00. The Williams & Wilkins Co., Mt. Royal and Guilford Avenues, Baltimore 2, Maryland, 1956.

THE SPINE; ANATOMICO-RADIOGRAPHIC STUDIES, DEVELOPMENT AND THE CERVICAL REGION. By Lee A. Hadley, M.D., Senior Attending Roentgenologist, Syracuse Memorial Hospital; Clinical Associate Professor of Public Health, New York State College of Medicine; Consultant in Radiology, Syracuse State Medical School, Syracuse, New York. Cloth. Price, $6.50. Pp. 156, with numerous illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

INTRAVENÖSE CHOLANGIOGRAPHIE; GRUNDLAGEN, TECHNIK, ERGEBNISSE. Von Priv.-Doz. Dr. Th. Hornykiewytsch, Giessen. Cloth, Price, DM 54.—. Pp. 159, with 110 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

RENAL ANGIOGRAPHY IN EXPERIMENTAL HYDRONEPHROSIS. Acta radiologica, Supplement 136. By Hans Idbohrn. Paper. Price, Sw. Kr. 20:-. Pp.85, with 27 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

ON PARALLAX AND VARIATIONS IN THE STEREOSCOPIC IMAGE; WITH A VIEW TO STEREOFLUOROSCOPY. Acta radiologica, Supplement 139. By Tauno Mannila. Paper. Price, Sw. Kr. 25:-. Pp. 107, with 40 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

MEDICAL EFFECTS OF THE ATOMIC BOMB IN JAPAN. Edited by Ashley W. Oughterson, Clinical Professor of Surgery, Yale University School of Medicine, and Attending Surgeon, Grace-New Haven Community Hospital; and Shields Warren, Pathologist, New England Deaconess Hospital, and Professor of Pathology, Harvard Medical School. Cloth. Price, $8.00. Pp. 477, with numerous figures. McGraw-Hill Book Company, Inc., 330 West 42nd Street, New York, New York, 1956.

BIOLOGY OF THE LABORATORY MOUSE. By the Staff of the Roscoe B. Jackson Memorial Laboratory; George D. Snell, Editor. Unabridged reprint of first 1941 edition. Cloth. Price, $6.00. Pp. 497, with 170 illustrations. Dover Publications, Inc., 920 Broadway, New York 10, New York, 1956.

DAS SKELETT DES KINDES; ENTWICKLUNG, BILDUNGSFEHLER UND ERKRANKUNGEN. Von Doz. Dr. W. Swoboda, Wien. Cloth. Price, DM 48.—. Pp. 184, with 199 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

TWENTIETH SEMIANNUAL REPORT OF THE ATOMIC ENERGY COMMISSION. Paper. Pp. 260, with illustrations and tables. United States Government Printing Office, Washington, D. C., 1956.

CANCER OF THE ESOPHAGUS AND THE STOMACH; A MONOGRAPH FOR THE PHYSICIAN. Sixth of a Series on the Early Recognition of Cancer. By Owen H. Wangensteen, M.D., Professor of Surgery and Head of Department of Surgery, University of Minnesota Medical School, Minneapolis, Minnesota. Second edition. Paper. Free. Pp. 144, with 60 figures and 9 tables. The American Cancer Society, Inc., 521 West 57th Street, New York 19, New York, 1956.

Vol. 76, No. 4

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Schulze, H.-E. Zur Röntgendiagnostik der intrakraniellen Epidermoide (sog. Cholesteatome). (The roentgen diagnosis of intracranial epidermoids [so-called cholesteatomas].) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, April, 1956, *84*, 440–446. (Address: Johannisallee 34, Leipzig-C 1, Germany.)

Eleven cases of intracranial epidermoids are described. While these growths comprise only from 0.2 to 1.1 per cent of all brain tumors, a specific diagnosis can be made by pneumoencephalography in a considerable number of them. Routine skull roentgenograms are not revealing except for the possible presence of secondary signs of increased intracranial pressure or calcification in the tumor. Cerebral arteriograms usually show only displacement of the vascular system by a space-occupying lesion. The so-called staining of the tumor is rare.

In 8 of the 11 cases pneumoencephalography revealed only the presence of a space-occupying lesion. In 3 cases a specific diagnosis of an epidermoid could be made because of the air staining of the tumor itself. The entrance of air into the substance of the tumor is caused by a rupture of the thin capsule of the tumor which allows air to penetrate the fissures of the growth, thus depicting the layers between the cholesterin filled spaces typical for this tumor.—*Hans W. Hefke, M.D.*

Leight, Ted F., Fincher, Edgar F., and Hall, Maxwell F., Jr. Evaluation of routine skull films in intracranial meningiomas. *Radiology*, April, 1956, *66*, 509–517. (Address: Dr. Leight, Emory University Hospital, Emory University, Atlanta, Ga.)

The meningioma is a slow growing, benign tumor of the arachnoid, which occurs with relative frequency in the intracranial cavity. It is an ideal lesion for study by the roentgen ray, since at least 50 per cent of all routine skull series are positive in its presence. These neoplasms may produce 1 or more of the following positive findings in the routine skull examination: (1) bony alterations, either productive or destructive; (2) vascular alterations; (3) tumor calcifications; (4) sellar alterations; and (5) pineal shift. Of these five classes, 3 are often characteristic of meningioma, namely, bone production, increased vascularity, and tumor calcification. These 3 are discussed in this paper.

In the presence of meningioma, either destructive or proliferative changes may be noted in the skull, although hyperostosis is more common and is estimated to occur in about 25 per cent of cases. Four different degrees of destructive process have been observed. These are: (a) pressure thinning of the inner table; (b) erosion of the inner table; (c) destruction of the inner table and erosion of the outer table; and (d) destruction of the inner and outer table.

Since meningioma is a highly vascular tumor, it is capable of causing changes in the vascular channels of the skull. These changes may be localized to the site of the tumor or may involve the vascular channels of the skull supplying the tumor. It has been pointed out by many authors, and is emphasized in this paper, that unilateral enlargement of vascular channels and foramen may occur as a normal variant, so that this finding is not sufficient for establishment of a diagnosis of a space-occupying lesion unless supported by appropriate clinical findings.

Meningiomas at times contain calcium. In Camp's series, 18 per cent produced roentgen evidence of calcification. He further states that the calcific deposits "have a characteristic roentgenographic appearance, which is imitated only by those occurring in the ependymomas." The calcium is within the psammoma bodies of the tumor, and produces a typical punctate appearance on the roentgenogram. The deposits may be widely scattered or so compact that they seem to be nearly homogeneous.

Monckton, in his series of cases, noted 3 different configurations of the calcium deposits. He described flocculent, linear, and wheel-like appearance, and considered the linear deposits to be within the tumor capsule.—*A. Ralph Watson, M.D.*

Neck and Chest

Endress, Z. F., and Schnell, F. R. Varicella pneumonitis. *Radiology*, May, 1956, *66*, 723–726. (Address: Z. F. Endress, 410 Pontiac State Bank Building, Pontiac, Mich.)

During the early part of the second week of illness, patients with chickenpox sometimes develop the serious complication of varicella virus pneumonitis. Eighteen such cases have been reported in the literature in the past fifteen years. Seven of these had a fatal outcome, with multiple organ system involvement in each. Autopsy findings were similar in all cases, there being disseminated areas of necrosis in epithelial tissues. The lungs showed patchy bronchopneumonia with confluent areas of consolidation.

Chest roentgenograms are reproduced in 12 of the case reports, but in only 2 was there follow up to complete clearing. The findings at the height of the illness were quite similar in all, varying in degree only. All 5 lobes were involved, with patchy areas of bronchopneumonia tending to coalesce in the more severe cases but favoring no particular portion of the lung fields. Early regression was rapid, paralleling the course of the skin lesions. Slow but steady improvement followed, and a normal condition was

reached within six months in the 2 cases followed for that length of time.

Clinically, severe dyspnea, cyanosis, and hemoptysis are commonly associated with the pneumonia. Nephritis, encephalitis, hepatitis, pericarditis, orchitis, and laryngitis may also be present.

The authors present a case (which they followed to complete clearing), since a search of the radiologic literature revealed only two similar cases reported.—*A. Ralph Watson, M.D.*

Kriss, Nathan. Chickenpox penumonia; a case report. *Radiology*, May, 1956, *66*, 727–729. (Address: 95 Highland Ave., Geneva, N. Y.)

Chickenpox pneumonia is a serious complication of the common varicella infection, occurring in approximately 8 out of every 1,000 cases. The incidence is much higher in adults with chickenpox, than in children. Usually it occurs two to five days after the rash has appeared.

A nonspecific diffuse miliary pattern is seen on the roentgenogram, which should be characteristic enough to distinguish from secondary bacterial consolidations, although both complications may co-exist. These findings slowly regress over a period of two and a half months, lagging far behind clinical improvement, and a residual fine mesh of interstitial thickening may remain.

A case report is included in which the initial chest roentgenogram showed a diffuse miliary pattern with a tendency to conglomeration in the mid and lower zones. This pattern initially was somewhat obscured by alveolar exudate. A roentgenogram three days later showed clearing peripherally, with less conglomeration, and more prominent stippling. Six days later, beading and coarse reticulation were the outstanding features, and it was not until nine weeks after admission that the nodular densities had disappeared, although some reticulation remained.—*Charles L. Stevenson, M.D.*

Heckmann, K. Der lokalisierte Pleuraerguss. (The localized pleural effusion.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1956, *84*, 176–184. (Address: Hohenstraufenstr. 7, München 13, Germany.)

In contrast to free pleural effusion, the circumscribed ones are diagnosed much less frequently. However, these localized effusions are as common as the free ones, but since they are usually much smaller, a special technique is necessary to find them. There are 5 types of such effusions: (1) encapsulated; (2) interlobar; (3) diaphragmatic; (4) mediastinal; and (5) apical. Hydrodynamic and pathologic interpretations as regards these types of pleural effusions need improvement. Zuppinger and Westermark described certain types of effusions occurring in the presence of atelectasis. One has to distinguish between the zone where the exudate occurs and the one where it is collected. The tendency of a pulmonary segment to diminish its volume determines the location and shape of the effusion. An example is given and illustrated by reproduction of 2 roentgenograms where a small exudate appeared to be encapsulated at the chest wall in deep inspiration, but proved to be free in expiration.

In other cases, the effusion is being sucked into the interlobar spaces. A similar situation exists in the diaphragmatic effusion, in which the costophrenic angle might appear clear and diaphragmatic motility might be maintained with fluid only demonstrable in the decubitus position, or, on the left, by the increased distance between the cardia of the stomach and the diaphragm. Occasionally, the exudate is collected in the upper mediastinum (one such case is described with reproduction of three roentgenograms. More frequent is an anterior mediastinal effusion which surrounds the heart like a cap and causes unsharpness of the cardiac contour (the authors calls this a paracardial effusion or a "halo-sign"). The unsharpness was formerly thought to be due to motion, which, however, with modern equipment no longer is of significance. Frequently, this unsharpness disappears in the supine position which also helps to differentiate it from pleuropericardial adhesions. This likewise is illustrated with several roentgenographic reproductions.

Another important location of hidden effusions are the apices. This is frequently confused with "thickened apical pleura." The presence of such an effusion can be proved by induction of a pneumothorax which is followed by disappearance of the apical density and collection of a small amount of fluid at the costophrenic angle.

The great practical importance of these small localized effusions lies in the fact that they indicate a collapse tendency of the adjacent lung segment. This, on further study, might reveal a hidden bronchial carcinoma, or a small circumscribed lesion of a different nature.—*Egon G. Wissing, M.D.*

Barry, William F., Jr. Infrapulmonary pleural effusion. *Radiology*, May, 1956, *66*, 740–743. (Address: 1022 Gloria Ave., Durham, N. C.)

Small amounts of pleural effusion are difficult to demonstrate on routine chest roentgenograms as there may exist as much as 500 cc. of pleural fluid in the infrapulmonary space, the portion of the pleural cavity between the diaphragm and the inferior surface of the lung, without it being evident on the routine films. The author presents a study concerning 7 patients with effusion in this infrapulmonary space.

During a twelve month period from February,

1953 through January, 1954, 52 cases of effusion were seen in the roentgenologic department of a 150-bed hospital. Seven of these had effusions in the location described above and are the basis of this paper. Such fluid collections are more difficult to detect in the right chest cavity than in the left, since the density of the liver is the same as that of the fluid. An elevated right diaphragm should always arouse suspicion that fluid is present and should suggest that further study is needed. On the left side, elevation and separation of what appears to be the diaphragm and the gastric bubble should bring this possibility to mind. Here, air or gas may be introduced into the stomach which will then aid in delineation of the lower margin of the diaphragm and will demonstrate the relative thickness of the fluid layer.

Fluoroscopically, fluid waves, propagated by the heart motion, may be observed along the upper border of the "elevated diaphragm" proceeding laterally from the mesial portion of the chest cavity. Also, by having the patient lean to the affected side, the fluid may be directed into the lateral costophrenic sulcus. Pneumoperitoneum will delineate the lower surface of the diaphragm and thus show the thickness of the layer of fluid. Another procedure which is probably the least difficult and the most productive of reliable results is that of obtaining lateral decubitus films with the suspected side down. In that position the fluid flows from the infrapulmonary space out along the dependent lateral chest wall.

The author feels that these infrapulmonary collections of fluid are much more frequent than the number of reports in the literature would indicate and that the frequency of discovery of this process is directly dependent on the index of suspicion of the radiologist and clinician.—*Donald N. Dysart, M.D.*

Keats, Theodore E., Kreis, Van Allen, and Simpson, Ellen. The roentgen manifestations of pulmonary hypertension in congenital heart disease. *Radiology*, May, 1956, *66*, 693–700. (Address: T. E. Keats, University of California Medical Center, San Francisco 22, Calif.)

Pulmonary hypertension can be quantitatively demonstrated at present only by cardiac catheterization. Roentgen criteria which are useful in recognizing this situation on routine chest films in acquired heart disease have recently been suggested by British investigators. This study was undertaken in order to determine whether these criteria can be applied also to the congenital heart lesions.

The criteria employed in these British studies were enlargement of the heart, enlargement of the right ventricle, prominence of the main pulmonary artery, prominence of the perihilar vessels, disproportion between the size of the enlarged central pulmonary arteries and narrowed peripheral pulmonary arteries, tortuosity of peripheral pulmonary arteries, and the presence of fine horizontal lines at the periphery of the lungs near the costophrenic angle (Kerley's lines). These British authors were able not only to detect the presence of pulmonary hypertension, but also to correlate the magnitude of the changes with the degree of hypertension. The Kerley's lines are thought to be due by some authors to engorged peripheral lymphatics or interstitial edema and by others to hemosiderin adjacent to and within the interlobular septa or fluid in the interlobular septa.

Case records and chest roentgenograms of 36 patients with congenital heart disease and proved pulmonary hypertension, demonstrated by cardiac catheterization, were reviewed. Films of 15 patients with the same types of heart disease but with normal pulmonary artery pressures were also reviewed. The congenital lesions involved in this study all resulted in a left-to-right shunt and consisted of ventricular septal defects, atrial septal defects, patent ductus arteriosus, and one unclassified septal defect.

Results of this study showed no relationship between the degree of hypertension and the type of congenital defect present. A correlation of the magnitude of pulmonary blood flow with size of the cardiac silhouette, right ventricle and the pulmonary artery was found, as an increase in pulmonary flow tended to result in an increase in size of the silhouette, right ventricle and the pulmonary artery. The only one of the above criteria used in determining the presence of pulmonary hypertension in acquired heart disease that was applicable in congenital heart disease, with a left-to-right shunt, was disproportionate narrowing of the peripheral pulmonary arteries as compared to the proximal arteries. This sign was present in 64 per cent of the cases studied, and consisted of a rather abrupt decrease in the caliber of the blood vessels usually adjacent to the dilated hilar portion of the artery.—*Donald N. Dysart, M.D.*

Abdomen

Richards, A. J. (Regina, Saskatchewan.) Lymphosarcoma of the stomach. *J. Canad. A. Radiologists*, Dec., 1955, *6*, 60–64.

An excellent review of the literature on gastric lymphosarcoma is given. At least 518 cases of the disease, primary in the stomach, have now been reported.

Between 1932 and 1954, 13 cases of primary gastric lymphosarcoma were found in Saskatchewan, representing an incidence of 0.75 per cent of all malignancies of the stomach. When all cases of lymphosarcoma with gastric involvement were considered, the incidence rose to 2.3 per cent. No racial predilection for the disease was found. Sex incidence revealed a male predominance of 3.4 to 1. There was a relatively greater incidence of the disease in the third decade, but almost 60 per cent of the cases were

in the fifty to eighty year age range. The latter was due to the relatively greater incidence of generalized lymphosarcoma in the older age groups. Many features of lymphosarcoma of the stomach simulate carcinoma so closely that differentiation is difficult. The usual roentgenologic findings are: (1) large polypoid or infiltrating defect out of proportion to the severity of the clinical picture; (2) enlarged rugal folds; (3) peristalsis and pliability not completely eliminated; and (4) ulcer usually deep and large when present.

The importance of operative and preoperative diagnosis is stressed by the fact that in the author's series of 13 primary cases, 4 are alive and well, one to five years postoperatively. This bears out the opinion that the prognosis is better than that in carcinoma of the stomach, and every effort should be made to afford these patients adequate roentgen therapy.—*Arch H. Hall, M.D.*

Gimes, B. Pharmakoradiologischer Beitrag zur Frage des sog. Magenschleimhautprolapses. (Pharmaco-roentgenologic observations on prolapse of the pyloric mucosa.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1956, *84*, 288–294. (Address: Üllöi út. 78/b., Budapest VIII, Hungary.)

The existence of a prolapse of the pyloric mucosa as a distinct pathologic entity is apparently not accepted by some continental radiologists including the author of this paper. The roentgen appearance described as prolapse is believed by the author to be caused by projection of the widened mucosal fold of the pyloric region into the base of the duodenal bulb. This widening of the mucosal folds is apparently due to increased autoplasty of the mucosa which can be caused by various factors as edema, inflammatory changes, tumors and mechanical factors.

A case is described of a fifty year old male with ulcer history and a typical roentgen appearance of prolapse of the pyloric mucosa accompanied by widening of the mucosal fold of the antrum.

Five minutes after subcutaneous injection of 5 mg. of morphine, the appearance suggesting prolapse subsided and the antral mucosal folds presented normal width. The usual effects of morphine (hyperperistalsis, prolonged patency of the pylorus and prolonged filling of the duodenal cap) were observed and an ulcer crater became visible in the prepyloric region on the lesser curvature. A control examination four months later showed again an appearance considered typical for prolapse which subsided after morphine.

A second case, that of a 74 year old male, was practically identical as far as roentgen appearance before and after morphine was concerned. The cause of the widening of the mucosal folds was in this case also a prepyloric ulcer.

The explanation for this effect of morphine is sought in Forsell's classic teaching about the two separate dynamic mechanisms of the gastric muscularis and the mucosal autoplasty.

This article contains interesting observations on a controversial subject and should stimulate further experiments along similar lines.—*Karol E. Matzinger, M.D.*

Lafebvre, J., Sauvegrain, J., Pellerin, D., Anguenot, G., and Bennet, J. Étude radiologique des sténoses duodénales par brides et volvulus chez le nourrisson et chez l'enfant. (A propos de 45 observations). (A radiologic study of duodenal stenosis in sucklings and infants as the result of adhesions and volvulus.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, Jan.-Feb., 1956, *37*, 1–11. (From: Service Central d'-Electroradiologie de l'Hôpital des Enfants Malades, Paris, France.)

The authors point out that duodenal stenosis in sucklings and infants is frequent. It follows embryologic variation in the development of intestine and peritoneum. The condition arises either from congenital peritoneal bands or from volvulus following anomalous rotation. A common mesentery is not the primary cause of duodenal stenosis but more or less apposition of loops which accompany this condition can give rise to severe stenosis.

The major clinical sign of duodenal stenosis is vomiting of bile. This is characteristic of most cases with the stenosis below the ampulla. When it is above the ampulla, the vomiting is still characteristic but contains no bile.

Roentgenologic study establishes the location of the stenosis and gives information regarding anomalies in rotation. The entire gastrointestinal tract should be examined including the colon. There is some correlation, which however is not complete, between the clinical and roentgenologic signs.—*Marcy L. Sussman, M.D.*

Poppel, Maxwell H., and Jacobson, Harold G. Roentgen aspects of the papilla of Vater. *Am. J. Digest. Dis.*, Feb., 1956, n.s. *1*, 49–57. (From: Departments of Radiology of New York University College of Medicine and Bellevue and Montefiore Hospitals, New York, N. Y.)

The author studied over one hundred autopsy and surgical specimens of the mid duodenum including the papilla of Vater to attempt to establish roentgen criteria of abnormality.

The average length of the papilla was 1.5 cm., average width 0.5 cm. These sizes varied from 3.0 cm. to 1.2 cm. in width by 1.2 cm. in depth to 0.1

cm. in length by 0.1 cm. in width by 0.1 cm. in depth. The papillae were consistently located on the medial wall, posteriorly, in the mid descending duodenum. The minor papillae were located in front of the major one at an average distance of 1.8 cm. Almost all were smaller than the major papillae but one was larger.

The points that should be noted in studying this area roentgenologically are size, position, irregularity in shape, abnormalities in the mucosal pattern in the peripapillary area, abnormal duodenal peristalsis or configuration, duodenal diverticula, and the presence of a palpable mass.—*G. L. Sackett, Jr., M.D.*

Glazenbrook, A. J., Mongeon, C., and Wong, E. The use of pro-banthine and of baridol in the visualization of the mucosal pattern of the small intestine. *Canad. M.A.J.*, Feb. 15, 1956, *74*, 280–285. (From: St. Boniface Hospital and Department of Medical Research, University of Manitoba, St. Boniface, Manitoba, Canada.)

The routine examination of the small intestine is is often fraught with difficulties due to basic physical properties of the barium used and the physiologic functions of the intestine. Discontinuity, flocculation, and bolus formation are the problems which the authors attempted to correct by the use of colloidally stabilized baridol and by the administration of the anticholinergic substance probanthine. Eleven medical students were the subjects for these experiments. Initial control studies using plain barium sulfate presented the inherent difficulties described above. The addition of probanthine hardly altered these effects. Undiluted baridol gave excellent views of the duodenum and jejunum in almost all cases but ileal detail was completely obscured by massing of the medium into solid columns, as well as, by discontinuity and bolus formation. When probanthine was given, in addition to baridol, there was a significant delay in small intestinal mobility, but about half of the subjects showed excellent images of the upper ileal segments. However, the best results were obtained when 7 ounces of baridol were mixed with 5 ounces of water and given one hour after a single dose of 15 mg. of probanthine by mouth.

The attempted diminution of water absorption in the small intestine by using probanthine is based upon the supposition that it would reduce both segmentation and activity of the villi. Although it would also inhibit peristalsis and slow the transit time, the net effect would be to reduce water absorption and aid in the maintenance of the fluidity of the barium meal. Visualization of the distal half of the ileum was unsatisfactory in the authors' series but they suggest that modifications of this technique might be used for this purpose.—*Arch H. Hall, M.D.*

Wolf, Bernard S., and Marshak, Richard H. Segmental infarction of the small bowel. *Radiology*, May, 1956, *66*, 701–707. (Address: B. S. Wolf, 1 East 100 St., New York 29, N. Y.)

Impairment of the blood supply to the intestinal tract produces a variety of changes which may be evident on roentgen studies. These circulatory disturbances may be secondary to strangulating obstruction, as a volvulus or strangulated hernia, or primary, as a result of occlusion of the veins and arteries of the intestine by thrombi or emboli. Most of these cases require immediate surgical intervention, but a small number will recover from this initial episode if the insult has involved only a short segment of the intestine and resulted in only partial devitalization instead of gangrene and perforation. In such an event, healing may occur and be followed by stenosis.

This paper describes the roentgen findings in a group of cases with small intestinal infarction which did not go on to complete recovery, but rather to segmental stenosis and obstruction. These findings consist of tubular narrowing of a segment of intestine no longer than 6 inches and usually no shorter than 3 inches, with effacement of the mucosal pattern but none of the characteristics of mucosal destruction and no evidence of nodular irregularity of the lumen or overhanging edges. The degree of narrowing is variable, depending on the progress of the disease. The transition from the dilated intestine proximal to the lesion to the normal distal intestine is smooth, concentric, and usually fairly abrupt. The differential diagnosis includes: Hodgkin's disease, metastatic carcinoma, lymphosarcoma, myosarcoma, adenocarcinoma, malignant carcinoid and myoma.

The authors discuss 4 cases, one due to a strangulated hernia, which resulted in only partial devitalization of the intestinal wall, another due to venous occlusion on the basis of thrombo-angiitis obliterans, and 2 in which the etiology could not be proved.—*Donald N. Dysart, M.D.*

White, Harvey. Meconium ileus: A new roentgen sign. *Radiology*, April, 1956, *66*, 567–571. (Address: Children's Memorial Hospital, 707 Fullerton Ave., Chicago 14, Ill.)

The author calls attention to a roentgen sign not previously described, which is believed to be diagnostic of uncomplicated meconium ileus. Six cases of meconium ileus, with classical signs of obstruction are reported. The same roentgenographic findings were present in all: many gas-filled dilated loops of intestine on the supine films and absence of fluid levels on the upright films. An additional 4 cases are reviewed, but are not reported in detail. They were complicated by the existence of volvulus, gangrene or peritonitis, but fibrocystic disease was found to be the primary disease.

The author concludes that the diagnosis of meconium ileus is justified whenever dilated loops of small intestine with no fluid levels are demonstrated on the upright abdominal roentgenogram of a newborn with clinical signs of intestinal obstruction.—*A. W. Sommer, M.D.*

Fleischner, Felix G., and Berenberg, Arnold L. Recurrent carcinoma of the colon at the site of the anastomosis; roentgen observations. *Radiology*, April, 1956, *66*, 540–547. (Address: Dr. Fleischner, Department of Radiology, Beth Israel Hospital, Boston 15, Mass.)

Recurrence of carcinoma—particularly of the sigmoid and, to a lesser degree, of the descending colon—at the site of the end-to-end anastomosis, or the ileocolostomy, has been seen frequently. This recurrence has been thought to be due to: (a) continuous growth of cancer tissue inadvertently left behind by the surgeon; (b) malignant degeneration of a neighboring polyp; (c) development of fresh carcinomatous changes from predisposed epithelium in the neighborhood of the original tumor.

A new concept is that these recurrences are due to a distinct pathologic process, i.e., implant metastasis, caused by implantation of cancer cells in the suture lines during cutting and suturing at the time of operation.

The roentgen changes at the site of the operation depend on the surgical technique, on the postoperative course, and on the time elapsed since operation. A permanent status was reached in one to six months after operation in uncomplicated cases. Patients with postoperative complications should be re-examined early, in order that a base line may be established for determining whether or not changes found later are due to the complications or to recurrence. In examining the site of anastomosis, every irregularity, any mass projecting into the intestinal lumen, even the size of a pea, is abnormal and should be recorded as such. Also, localized lack of pliability of the wall or gross irregularity of mucosal relief should be considered abnormal. Constrictions caused by inverted, sutures, granuloma, spasm, and edema may be found in patients examined after a longer interval. In most instances, the constrictions were bilateral or circular, but when they were unilateral, recurrent carcinoma was much more likely.

The first postoperative roentgen examination should be done, as a general rule, one or two months after surgery in the patients with uncomplicated postoperative course. Repeated examinations should then be done at variable intervals, always scrutinizing the site of anastomosis carefully and recording the findings with spot films and routine roentgenograms. Thus, the early diagnosis of a local recurrence is possible by having a means for comparison of later studies with the early postoperative findings.

Eleven case studies are presented along with several roentgenographic reproductions illustrating these various findings.—*Donald N. Dysart, M.D.*

Westing, S. W. A note on barium enema examination of incontinent patients. *Radiology*, April, 1956, *66*, 582–584. (Address: 43 Arnold Way, West Hartford 7, Conn.)

A method for the barium enema examination of patients whose anal sphincter control is so reduced that retention of the enema cannot be obtained even by the use of the occluding rectal catheter is described. The patient is placed upon a bedpan and the enema flow kept up until the barium suspension reaches the cecum or terminal ileum. This is usually accomplished before the amount expelled fills the bedpan to capacity. The metal bedpan interferes surprisingly little with the visibility, particularly if an aluminum bedpan is used. After the urge to evacuate has subsided the bedpan is removed and the patient can be rotated into any of the positions desired for proper fluoroscoping and filming.—*Harold C. Hamilton, M.D.*

Gaebel, E. Das negative oder kontrastschwache Cholezystogramm und die Diagnose der Cholezystitis. (The weak or absent opacification of the gallbladder and the diagnosis of cholecystitis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1956, *84*, 295–304. (From: Strahleninstitut der Allgemeinen Ortskrankenkasse, Köln, Germany.)

The author discusses the various causes of poor or absent opacification of the gallbladder after oral cholecystography, dividing them into three groups: (1) those preventing the contrast medium from reaching the liver, *i.e.*, mainly lesions of the digestive tract; (2) hepatic insufficiency; and (3) lesions of the gallbladder and biliary ducts.

A complete clinical evaluation of each case is necessary to exclude those causes of nonopacification falling into the first two groups. All patients with poor or absent opacification after an oral dose are examined with cholografin intravenously. If this shows good opacification, poor concentration of the contrast substance given orally was due to a lesion of the gallbladder wall. If both the oral and intravenous examinations fail to opacify the gallbladder while contrast material containing bile is visible in the common duct and the small intestine, an obstruction of the cystic duct is present preventing the contrast medium from entering the gallbladder. If faint opacification after both methods is present, partial obstruction of the cystic duct with cholecystitis is its cause. If no contrast medium containing bile is visible in the biliary ducts and in the intestine and the gallbladder is not opacified, hepatic insufficiency is present.

The poorly and occasionally also the well opaci-

fied gallbladder which is tender on palpation and fails to contract after cholecystokinetic meal is suspect of cholecystitis. A normal cholecystogram does not exclude incipient and localized cholecystitis.

Two authors are quoted, one claiming that 21.8 per cent of his cases where cholecystitis without stones was found showed a normal cholecystogram while the second gives a percentage of 53.—*Karol E. Matzinger, M.D.*

Waller, Robert D., and Roberts, S. M. Cholecystographic visualization of the Rokitansky-Aschoff sinuses: Report of a case and review of the literature. *South M. J.*, March, 1956, *49*, 221–225. (From: Veterans Administration Hospital, Augusta, Ga.)

The authors point out that, although diverticulosis of the gallbladder was known to surgeons and anatomo-pathologists since the seventeenth century, it was not until 1948 that the first reports on the roentgenologic visualization of the diverticula by cholecystography appeared in the literature. They were able to collect only 16 cases of chronic cholecystitis with Rokitansky-Aschoff sinuses diagnosed roentgenographically. To these they add their own case.

A rather complete report of a forty year old white man who had abdominal distress for several years is given with a review of the 16 cases available in the literature. Also, there is a discussion of the pathology and possible etiologic factors causing this condition.

A review of the literature with reference to roentgenographic demonstration of Rokitansky-Aschoff sinuses was undertaken. The term is applied to the outpouchings or herniations of the mucosa into and through the muscularis of the gallbaldder, some of which communicate with the lumen by epithelial lined ducts, others, having large openings, and still others with no communication.

The roentgenographic findings are rather consistent. Demonstration is somewhat rare, due to the inability of most diseased gallbladders to concentrate the contrast medium. When visualization does occur, the findings are as follows: (1) concentric distribution of the contrast medium seen approximately one millimeter from the contrasted gallbladder lumen; (2) abnormally shaped gallbladder shadow usually consisting of a "purse-string" narrowing near the neck of the gallbladder; (3) poor or delayed emptying; (4) lithiasis; and (5) mulberry appearance en face.

Of the 17 cases reported, 13 were proven pathologically as chronic cholecystitis with Rokitansky-Aschoff sinuses. The sinuses varied in size and number and measured up to 5 mm. in diameter.—*Robert D. Moreton, M.D.*

Mandel, William, Gaines, L. M., Jr., and Marilley, R. J., Jr. Evaluation of oral cholecystography in liver disease. *A.M.A. Arch. Int. Med.*, March, 1956, *97*, 335–339. (From: Medical Department, 3345 U.S.A.F. Hospital, Chanute Air Force Base, Ill.)

It has been pointed out that gallbladder visualization by oral route depends on an adequate blood level of contrast medium following its absorption, its removal and excretion by the liver, and its entrance into and concentration by the gallbladder. It has been frequently stated that cholecystography is contraindicated in liver disease with jaundice, for one cannot determine whether the failure of visualization is due to liver or to gallbladder disease. This study is to correlate the serum bilirubin level, the retention of sulfobromophthalein contrast medium and gallbladder visualization in 40 patients with varying degrees of liver disease and attempt to clarify this problem.

Tests of liver function and oral cholecystography were performed the same morning. When the gallbladder did not visualize, the determination was usually repeated within one week using a double dose of telepaque. Occasionally 12 tablets (or a double dose) were given the first time. The results of these tests are presented in 5 tables.

It was found that the presence of liver disease with jaundice is not a contraindication to cholecystography. It is the degree, and not the type of hepatic disease, which determines whether the liver can extract the contrast medium from the blood stream and transfer it to the biliary tract. It was concluded that though rigid rules cannot be established, a sulfobromophthalein retention below 20 per cent and a total serum bilirubin value lower than 5 mg. per 100 ml. should not interfere with gallbladder visualization. Sulfobromophthalein levels from 20 per cent to 30 per cent and serum bilirubin values from 5 to 10 mg. per 100 ml. may, or may not, interfere with gallbladder visualization. Values above these levels usually interfere with good gallbladder filling.

Serial studies with a double dose of contrast medium are recommended by the authors when the gallbladder does not visualize. If visualization does not occur as the liver function tests return toward normal, the likely cause is extrahepatic disease. The failure of gallbladder visualization in mild or moderate hepatic disease suggests an extrahepatic cause for nonvisualization. Used in this fashion, oral cholecystography may be helpful in differentiating extrahepatic from intrahepatic causes of jaundice.

It is further pointed out that unsuspected cholelithiasis may be found as the cause for or concomitantly with the liver disease, if cholecystography is performed more frequently in the jaundiced patient. —*Robert D. Moreton, M.D.*

Glazenbrook, A. J., and Hastings-James, Richard. Cholografin methylglucamine. *Canad. M.A.J.*, Feb. 15, 1956, *74*, 262–267.

(From: St. Boniface Hospital, St. Boniface, Manitoba, Canada.)

The authors report experiences in using a recently developed medium for intravenous cholangiography on 29 patients. The substance, cholografin methylglucamine, is equivalent to a 40 per cent solution of the sodium salt solution known as cholografin. Theoretically, an equal degree of radiopacity should be obtained with one-half of the quantity of the new substance, as compared with the usual quantity of cholografin. This advantage appeared to be outweighed by the increased number of side-effects produced. These included coughing and throat "tightness," facial flushing and conjunctival injection, momentary nausea, abdominal discomfort, and biliary colic. Ten of the 29 patients experienced 1 or more of these side-effects, many of which could be alleviated by a slower rate of injection. The slower rate of 20 cc. in three minutes denied one of the advantages looked for in a more concentrated preparation, namely of getting a higher blood level of cholografin, and therefore, a more rapid and heavy concentration in the bile.

The use of cholografin methylglucamine was definitely advantageous in several diagnostic problems, in which ordinary cholografin failed to yield satisfactory common duct visualization. The new medium also opacified the gallbladder to a degree comparable to a double dose of telepaque in certain instances. It would seem, therefore, that the radiologist has a welcome addition to his diagnostic armamentarium which may supplement though not replace cholografin in intravenous cholangiography.—*Arch H. Hall, M.D.*

Mintz, A. Aaron, Church, Gaylord, and Adams, Erie D. Relationship of cholelithiasis to sickle cell crises. *South M. J.*, March, 1956, *49*, 205–209. (From: Department of Pediatrics, Baylor University College of Medicine; Pediatric Out-Patient Service, Jefferson Davis Hospital; and Pediatric Out-Patient Service, Hermann Hospital, Houston, Tex.)

This is a report of routine cholecystography in 21 patients with sickle cell anemia. An incidence of 10 per cent of cholelithiasis was noted which corresponds to that of another clinical study and thus clinically confirms the high incidence of cholelithiasis with sickle cell anemia as found in autopsy studies.

The occurrence of abdominal pain in patients with sickle cell anemia is not satisfactorily explained. The incidence and importance of gallbladder disease in such patients has not received proper emphasis in view of the significant incidence of biliary calculi. Case reports of the 2 patients with gallstones are presented, one a ten year old and the other a fourteen year old Negro boy, each of whom had abdominal pain.

Increased destruction of red blood cells is a characteristic feature of sickle cell anemia as in all hemolytic anemias. Upon disintegration of the red blood cells, bilirubin is formed from the liberated hemoglobin. It is believed this excess of bilirubin favors aseptic precipitation of pigment in the biliary tract with resultant formation of stones.—*John F. Weigen, M.D.*

Gynecology and Obstetrics

Camiel, Mortimer R., and Aaron, Jules B. The gas or vacuum phenomenon in the pubic symphysis during pregnancy. *Radiology*, April, 1956, *66*, 548–552. (Address: Dr. Camiel, 149 New York Ave., Brooklyn 16, N. Y.)

The authors present a discussion of gas phenomenon in the pubic symphysis in pregnancy. Their conclusions are based on a study of the records and roentgenograms of 100 unselected cases.

It was found that, of the 100 pubic symphyses examined roentgenographically late in pregnancy, 30 showed a demonstrable cleft or clefts represented by a gas or vacuum shadow. This phenomenon was found to be a frequent, but not troublesome concomitant of the pelvic relaxation incidents to pregnancy, but whether it ever represents a physiologic or a pathologic mechanism is undetermined. The possibility of a traumatic factor is suggested, however.

It appears that the major influence on the joint is hormonal rather than mechanical, as the fissure shadow has been reported early in the course of pregnancy and its incidence is as high in breech as in cephalic presentations. It apparently bears no demonstrable relation to the outcome of labor.—*A. Ralph Watson, M.D.*

Rummel, A. Die röntgenologische Diagnose der Placenta praevia. (The roentgenologic diagnosis of placenta previa.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1956, *84*, 234–240. (Address: Univ.-Frauenklinik, J.-Schneider-Str. 4, Würzburg, Germany.)

The visualization of the placenta is of clinical and theoretical interest. In a previous paper, the author has described his method, which consists of the use of a modified Reid-aluminum filter without the use of contrast media. The present paper reports the results in diagnosis of placenta previa.

Placenta previa occurs once in 200 to 600 deliveries. The high mortality in home deliveries emphasizes the importance of correct diagnosis for mother and child. In vaginal examination, bleeding is frequent, indicating the necessity of a non-dangerous type of examination. The author combines the soft-tissue technique with the "displacement method" described by Ball and Golden. Films are exposed at

48-inch distance with 75-80 kv. and 200–300 ma. For details of the technique the original paper should be consulted. Fear of radiation damage should not interfere with this examination since the author has calculated a dose of 0.3 r per exposure.

There are three excellent roentgenographic reproductions.—*Egon G. Wissing, M.D.*

Schwarz, Gerhart S. An orthometric radiograph for obstetrical roentgenometry. *Radiology*, May, 1956, *66*, 753–761. (Address: 622 W. 168th St., New York 32, N. Y.)

A simple method which permits precision measurements of the bispinous and all other transverse pelvic diameters is described and the name "orthometric radiography" given to this method. With the patient supine on the roentgen table and the buttocks resting on a cassette with a built-in cross-hatched 7:1 grid, an exposure is made centered over the medial aspect of the right hip using a rectangular cone which will cover an area approximately 10×20 cm. on the film. The tube is then shifted transversely across the table by 10 cm., and a second exposure is made on the same film with the patient and cassette in their original position. A roentgenogram of the pelvis, consisting of two contiguous rectangles, is thus obtained. The only major factor controlling the presence or absence of roentgenographic distortion is the difference between the length of the tube shift and the actual length of the dimension to be measured. If this difference is small, the pelvic diameter will be depicted on the film in actual size with a high degree of accuracy.

A study of 350 consecutive roentgen cephalopelvimetries revealed that the most frequent cause of error was inaccurate measurement of the bispinous diameter of the pelvis. The determination of the bispinous diameter by orthometric radiography proved to be clinically more accurate than the conventional pelvimetric roentgenography, producing a better correlation between measurements and obstetrical outcome. This method may make it possible to diagnose high disproportion in clinically doubtful cases with an accuracy of more than 90 per cent.—*Harold C. Hamilton, M.D.*

Osmond, John D., Jr., and Rotter, Carl W. Unusual uterine enlargement caused by bleeding due to hypofibrinogenemia associated with fetal death. *Radiology*, April, 1956, *66*, 553–555. (Address: Dr. Osmond, 10515 Carnegie Ave., Cleveland 6, Ohio.)

Hypofibrinogenemia due to fetal death is an unusual cause of uterine bleeding and enlargement. The case presented differs from those previously reported, in that the fetus died from severe hemorrhage following placental separation, rather than Rh incompatibility.

Reid, *et al.* believe that hypofibrinogenemia in pregnancy can occur with long standing uterine retention of a dead fetus, amniotic fluid embolism, or severe premature separation of the placenta. It is thought that the defibrination of blood that occurs is due to an increased utilization or destruction of fibrinogen, secondary to intravascular coagulation caused by the escape of a coagulant, possibly thromboplastin, from the uterus into the maternal circulation. This defibrinating agent supposedly is derived from autolysis of decidua or placental tissue.

The diagnosis is proven by the demonstration of a low fibrinogen level, and it should be strongly suspected in any patient with a retained fetus who develops bleeding tendencies. An abnormally low sedimentation rate is usually found. Commercial fibrinogen should be given intravenously.—*Charles L. Stevenson, M.D.*

Cusmano, Joseph V. Dermoid cysts of the ovary: roentgen features. *Radiology*, May, 1956, *66*, 719–722. (Address: Englewood Hospital, Englewood, N. J.)

The roentgenologic findings in 35 surgically proved dermoid cysts of the ovary are presented. The cyst was evident roentgenographically in all but 5 patients of this group. Three of these 5 patients were pregnant at term, and the overlying fetal and maternal shadows probably obscured the lesion. In the other 2, obvious calcific elements were absent and poor technique and overlying gas and fecal material were believed responsible for nonvisualization.

The most consistent roentgenographic finding was a radiolucent area due to the fatty contents of the cyst which was seen in 25 instances. In many of the patients, the only finding was a rounded area of radiolucency limited by a well-defined wall 1 to 3 mm. in thickness. Calcific elements were identified in 17 cases. Most frequently, these were tooth-like structures, but in several patients the calcific material occurred in the form of single or multiple structureless masses bearing only a faint resemblance to teeth. In 1 case, thin calcific plaques outlined the entire cyst wall.—*Harold C. Hamilton, M.D.*

Genitourinary System

St. Martin, Eugene C., Campbell, James H., and Pesquier, Claude M. Cystography in children. *J. Urol.*, Jan., 1956, *75*, 151–159. (Address: Dr. St. Martin, 940 Margaret Place, Shreveport, La.)

This study of 60 children, indicates that cystography is an invaluable method which must be added to the usual procedures of excretory and retrograde pyelography and of cystourethroscopy in the detection of vesical dysfunction.

The authors show that it is not possible to predict existing back pressure from the cystoscopic appear-

ance of the ureteral orifice, the degree of bladder neck obstruction or the nature of the trabeculation. Furthermore, of 14 patients with marked reflux, only 3 were considered to have abnormal excretory urograms.

The procedure of cystography must be adapted somewhat to the individual patient. After voiding the child is catheterized for residual urine. The bladder is then filled by gravity, but not overdistended and a film is exposed. The catheter is removed and whenever possible a film is exposed during voiding. This may be either in the erect or horizontal position. Immediately after voiding a third film is exposed, and if necessary subsequent films are made as indicated. These films should include the kidney area and bladder. The authors do not feel that delayed cystography is necessary in every patient. By the use of this procedure vesico-ureteral reflux has been demonstrated in 50 per cent more patients than by other methods of study. In 74 "new" consecutive patients there was evidence of reflux in 10 or 13.5 per cent.

In conclusion the authors state "investigation of the pediatric urological problem is incomplete without cystography."—*George W. Chamberlin, M.D.*

Fetter, Theodore R., and Warren, Kenneth C. Congenital urinary tract obstructions in children. *J. Urol.*, Feb., 1956, *75*, 173–189. (Address: Dr. Fetter, 255 S. 17th St., Philadelphia 3, Pa.)

During the five year period 1950 to 1954, 369 pediatric urologic cases were studied. The various types of congenital obstruction are tabulated and representative cases are discussed in detail. Of 182 operative procedures, 36 were on the upper tract and 146 on the lower tract. Presenting symptoms are divided into 4 categories namely; (1) recurrent urinary tract infection; (2) obstruction with resulting pain either in the kidney or bladder; (3) the chronically ill child with anemia and symptoms referable to the gastrointestinal and respiratory tracts; and (4) absence of symptoms, the anomaly being an incidental finding.

In the majority of instances where defects exist in the external genitalia, the upper urinary tract is free of gross disease. However, those patients with multiple obstructive lesions are a formidable problem requiring careful evaluation of all diagnostic possibilities, diligent application of therapeutic principles and particular emphasis upon extended follow-up examinations. Attention to these factors and improvement in chemotherapeutic agents has already resulted in an increased salvage rate among these children.—*George W. Chamberlin, M.D.*

Nicolai, Charles H. Miokon, a new intravenous urographic medium. *J. Urol.*, April, 1956, *75*, 758–760. (From: Department of Surgery, Division of Urology, Washington University School of Medicine, St. Louis, Mo.)

Miokon is a new intravenous urographic medium containing 59.3 per cent of iodine and supplied in a 50 per cent solution of the sodium salt. The usual dose for adults is 30 cc. and for children proportionate to the body weight. Of 1,500 patients examined, 539 showed excellent quality urograms, 647 had good urograms, 264 had fair urograms, and 46 had poor urograms. In 4, no visualization was obtained. In the same number of patients, approximately 3.3 per cent had nausea, 1.8 per cent had nausea and vomiting and 9.6 per cent had one or more side reactions to the drug. None of the patients in this series had any severe side reactions, but transient hypotension occurred in 3 patients. This was severe enough to require supportive therapy.

The medium was also used in 30 translumbar aortographies with good results.—*George W. Chamberlin, M.D.*

Nesbit, Reed M. Diagnosis of intermittent hydronephrosis: importance of pyelography during episodes of pain. *J. Urol.*, May, 1956, *75*, 767–771. (From: Section of Urology, Department of Surgery, University of Michigan Medical School, Ann Arbor, Mich.)

The causes of intermittent pain in the upper abdominal quadrants or in the kidney region may be obscure or impossible to detect, unless the patient is examined during an attack. In this article the author presents 5 case histories and pyelographic findings to show the diagnostic importance of roentgenographic studies during the acute attack. He was able to demonstrate pronounced degrees of hydronephrosis during the acute attack which was not demonstrable at the time when the patient was not having pain. This emphasizes the great resiliency of the musculature of the upper urinary tract and that it can return to a normal, or almost normal, appearance when the obstruction has been compensated for. Surgical procedures have shown that urethral obstruction may be produced intermittently by aberrant vessels or congenital bands partially obstructing the ureters. Postural changes have also been shown to cause some degrees of hydronephrosis in a few instances.—*George W. Chamberlin, M.D.*

Secrétan, Michel. (Lausanne, Switzerland.) Les accidents au cours des urographies intraveineuses. (Accidents during intravenous urography.) *J. Belge de radiol.*, 1956, *39*, 76–87.

Some accidents following urography are preventable. Renal insufficiency is responsible for most delayed deaths. Certain liver affections, malignant hypertension, cardiac insufficiency, fever and cachexia are contraindications. In hyperthyroidism, it is well

to be prudent, particularly in the presence of iodine hypersensitivity and allergic states such as asthma and hay fever. Allergy is responsible ordinarily for immediate reactions.

The author groups the urographic media and refers to the hypothesis of Gautot that those containing ethalolamine have most often been associated with severe accidents. Although it has been established that a small amount of free iodine is present in these media, it is unlikely to be the direct cause of the reaction, but iodine idiosyncrasy remains a possibility.

The various tests of sensitivity are unreliable and misleading. It is only with allergic patients that sensitivity tests may be useful. The immediate reactions are treated with antihistamines given either intramuscularly or by slow intravenous injection. These substances should always be at hand. They should be used prophylactically when an allergic history is elicited. They should also be administered when there have been several previous urographic examinations and when one of the group of the aminoethanal salts of the diiodo-pyridone N acetic acid is used.—*Marcy L. Sussman, M.D.*

EVANS, JOHN A., and ROSS, WAYNE D. Renal papillary necrosis. *Radiology*, April, 1956, *66*, 502–508. (Address: Dr. Evans, New York Hospital, 525 E. 68th St., New York 21, N. Y.)

Renal papillary necrosis is an uncommon condition, usually found in diabetic patients, although it may occur in non-diabetics. Since this condition is not well known, the authors have reported a single case which was diagnosed roentgenographically during life.

The roentgenographic findings in this entity are the result of destruction of the papillae and or the associated inflammatory process. The diagnosis is therefore dependent on the phase of the disease in which the examination is performed. Roentgen findings in the various stages of the condition are as follows: (1) delay in or complete lack of function, most commonly unilateral. This is probably the earliest roentgen finding; (2) mild to moderate dilatation of the collecting system. This may be limited to a single calyx, a group of calyces, or the entire collecting system; (3) a mottled, moth-eaten appearance of the fornices. This results from necrosis and sloughing of the renal papillae; (4) gross filling defects throughout the calyces and pelvis, representing necrotic slough and blood clots; (5) radiolucent halos rimming dilated calyces. These halos are called by the authors "ring shadows," and probably represent defects left by a sequestrated papilla. They occur in an advanced stage of the disease; and (6) extravasation of the contrast material into the renal cortex. This is seen in advanced cases with peripheral necrosis and cavitation.

The diseases to be considered in the roentgenographic differential diagnosis are renal tuberculosis, renal tumor, renal calculus associated with colic, and acute pyelonephritis without necrosis.

In spite of the high mortality in patients with renal papillary necrosis, the correct antemortem diagnosis is of more than academic interest, since it is a condition which develops suddenly and progresses rapidly, making early and aggressive treatment of extreme importance. It is furthermore stated that the use of sulfonamide drugs in the treatment of this disease is hazardous, since these compounds have a tendency to precipitate in the renal tubules under conditions of urinary stasis or acid-base imbalance.—*A. Ralph Watson, M.D.*

FOORD, R. D., NABARRO, J. D. N., and RICHES, E. W. Diabetic pneumaturia. *Brit. M. J.*, Feb. 25, 1956, *1*, 433–434. (From: The Middlesex Hospital, London, England.)

Pneumaturia is an uncommon finding. If instrumental introduction of air can be excluded it is usually taken to indicate the presence of a fistula between the bladder and intestine or vagina. It is pointed out that the possibility of pneumaturia as the result of infection must also be considered, especially in diabetic patients. The authors have been able to trace the reports of only 2 cases of diabetic pneumaturia in the British literature. These cases are reported in some detail.

It is found that primary pneumaturia—that is, pneumaturia occurring in the absence of instrumentation or a bladder fistula—is more common in diabetics than in nondiabetics. It is associated with infection of the urinary tract, usually due to E. coli. Cystitis and pyelitis are common complications of diabetes and the authors feel that it is surprising that pneumaturia is not encountered more often. In many of the reported cases it has been associated with obstruction of the lower urinary tract or residual urine due to a diabetic neuropathy or vesical diverticulum. It has been suggested that pneumaturia, in some cases, may be the result of an infection with a strain of organism that produces an unusual amount of gas. The organism from the first case of the authors was shown to produce gas when grown in a broth containing glucose, but not in excessive amount.

The authors point out that when a patient has pneumaturia the possibility of diabetes should always be considered, and to exclude this it is not sufficient just to test the urine for sugar. In these patients the sugar from the urine may be removed completely by the bacterial action and a negative test obtained. Blood sugar estimates are required, or repeated urine testing after the infection has been controlled. The condition is probably much more common than published reports suggest.

The pneumaturia disappears if the glycosuria is controlled by suitable diabetic management and

the infection is eliminated by antibiotics or chemotherapy, combined when necessary, with relief of the obstruction of the urinary tract.—*Robert D. Moreton, M.D.*

Zatzkin, Herbert R. Hemochromatosis with diabetes, associated with cystitis emphysematosa. *Radiology*, May, 1956, *66*, 744–746. (Address: Meadowbrook Hospital, P. O. Box 108, Hempstead, N. Y.)

Cystitis emphysematosa and hemochromatosis with diabetes are rare entities, and their co-existence in a single individual is most unusual. A case report of such a circumstance is presented, in which roentgenographic studies were of definite help in making the diagnosis.

The salient roentgenographic findings were an unusually radiopaque liver shadow and transient gas in the wall of the urinary bladder. Liver cirrhosis and diabetes were substantiated clinically and confirmed at autopsy. Hemochromatosis was verified as the underlying cause.

Hemochromatosis is due to a disturbance in iron pigment metabolism, resulting in excessive deposits of hemosiderin and melanin in the skin and viscera, especially the liver and pancreas. The underlying cause is unknown, and it usually is seen in males past middle age. The associated fibrosis in the liver and pancreas may result in cirrhosis and diabetes. Exogenous hemochromatosis also occurs following repeated transfusions. Liver carcinoma has been reported to occur terminally in 7 per cent of patients with hemochromatosis. Increased radiopacity of the liver may be identified, and if previous thorotrast exposure can be ruled out, hemochromatosis is the logical diagnosis.

Transient gaseous infiltration of the bladder wall is due to an inflammatory condition, probably associated with bacterial invasion. It is seen most often in patients with diabetes. The interstitial air outlining the bladder produces a characteristic roentgenographic appearance.—*Charles L. Stevenson, M.D.*

Beneventi, Francis A., and Marshall, Victor F. Some studies of urinary incontinence in men. *J. Urol.*, Feb., 1956, *75*, 273–284. (Address: Dr. Beneventi, 65 E. 76th St., New York, N. Y.)

The first part of this paper consists of detailed histologic studies of the limits and course of the external sphincter of the urethra, as observed by serial sections of the tissues from the membranous urethra to the base of the bladder in 4 males aged seven, twelve, nineteen and forty years. In all 4 of these normal specimens a thick circular mass of striated muscle was found around the membranous urethra and prostatic apex. This muscle also formed the prostatic capsule in the apical portion and on the dorsum of the prostate the muscle continued much farther. It appeared that the extension of this striped muscle toward the bladder failed to reach the outlet by progressively greater distances as the age of the subjects increased so that the normal enlarging of the prostate with age could account for this anatomic variation in the specimens which were studied.

The second portion of the paper is concerned with roentgenographic observations of sphincteric actions of the bladder. By the use of rapid roentgenographic exposures or cineroentgenography, studies were made during voiding and resting in the normal and abnormal subjects. These studies indicated that the usual abrupt elevation of the bladder base on voluntary interrupting the stream was not essential for perfect continence. A few observations suggest that mobility of the levator ani muscle may be of some importance in maintaining excellent control. The external sphincter of continent men moved upward and downward for an inch, more or less, while the subject voluntarily started and stopped the urinary stream. This observation does not fit the popular conception of a sphincter inside a fixed triangular ligament. The authors reason that the bladder outlet changes in thickness and might have an action similar to that of a Chinese woven finger cuff. Inactivation of the external sphincter of continent men did not cause gross incontinence. The observations in this paper are designed to help clarify a physiologic action which is rather poorly understood at the present time.—*George W. Chamberlin, M.D.*

Nervous System

O'Connell, John E. A. Involvement of the spinal cord by intervertebral disk protrusions. *Brit. J. Surg.*, Nov., 1955, *43*, 225–247. (From: St. Bartholomew's Hospital, London, England.)

The paper presents a detailed study of protrusions of the intervertebral disks affecting the spinal cord proper. It is emphasized that, by definition the cases are limited to those in which a mass of disk tissue has escaped into the spinal canal. The more common lateral protrusion with impingement upon a spinal nerve is excluded, as are cases of spondylosis.

Fourteen such cases are presented, all of which were treated surgically; 8 occurred in the cervical spine, 4 in the thoracic spine, and 2 in the lumbar spine. In these cases, acute trauma did not play a significant part. Two additional cases are presented in which injury to the cord was felt to be the result of protrusion of an intervertebral disk against the cord as a result of an acute trauma.

These 14 cases are analyzed in such detail, under the following headings, so as to require that the original paper be read: age, sex, etiology, duration of illness, symptomatology, physical findings, accessory

methods of investigation, and surgical treatment and findings at operation.—*John F. Weigen, M.D.*

Skeletal System

Edinger, A., Gajewski, H., and Gepp, H. Röntgen-Ganzaufnahmen der Wirbelsäule. (Roentgenograms of the entire spine on a single film.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1956, *84*, 365–371. (Address: A. Edinger, Königstr. 35, Stuttgart-N, Germany.)

Following the trend of widening the scope of roentgenologic examination of the spine to include functional disturbances, the authors have developed a new method for upright roentgenography of the entire spine on a single film. The details of the procedure are the following:

To reduce distortion and ensure good detail, a distance of 3 meters (10 feet) and a rotating anode tube with a focal spot of 1.2 by 1.2 or 2 by 2 mm. are used. The exposure is 300 to 500 mas. A special upright cassette holder equiped with a moving grid measuring 40 by 100 cm. (approximately $15\frac{3}{4}$ by 39 inches) was designed provided with supports which help immobilize the patient during the relatively long exposure (3 to 5 sec.). To compensate for the wide variation in thickness between the cervical and lumbar segments and avoid overexposure of the thinner parts, experiments were carried out with wedge filters. These were found not entirely satisfactory as they increased the penetration of the beam while reducing its intensity which decreased contrast. Subsequently a rotating shutter placed between the anode and the patient was developed which cuts off radiation to the cervical and, to a lesser extent, the dorsal spine during part of the exposure and so ensures a film of uniform density. Shutters of various shapes adapted to the body type of the individual patient are used. The cassette measures 30 by 90 cm. (approximately 12 by 36 inches). A special film holder was designed which permits development of the films in tanks of ordinary size.

Photographs of the apparatus which is manufactured by Siemens are reproduced, as well as films showing the entire spine and pelvis in the anteroposterior and lateral projections with good bony detail and uniform density. This device should be of interest to orthopedic surgeons and radiologists dealing frequently with deformities of the spine.—*Karol E. Matzinger, M.D.*

Höffken, W. Das Wirbel-Asoma (Eine seltene Wirbelmissbildung). (The vertebra-asoma [a rare congenital malformation of the vertebra].) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, April, 1956, *84*, 483–487. (From: Röntgen- und Lichtinstitut der Städtischen Krankenanstalt, Köln-Merheim, Germany.)

Non-development of the body of a vertebra is called asoma. Absence of ossification of an existing cartilaginous anlage is not the same, although it also leads to an apparent absence of the body by compression of the persisting cartilage. In an asoma the posterior arch is fused anteriorly, such is not the case in the instance of presence of a cartilaginous vertebral body.

It is necessary to obtain a roentgenogram of the entire spine in order to determine definitely the numbers of the vertebral bodies and the corresponding posterior arches. Laminagraphy can be of considerable help in demonstrating this rare anomaly.—*Hans W. Hefke, M.D.*

Lusted, Lee B., and Pickering, Donald E. The hypothyroid infant and child; the role of roentgen evaluation in therapy. *Radiology*, May, 1956, *66*, 708–718. (Address: L. B. Lusted, University of California Medical Center, San Francisco 22, Calif.)

Roentgenographic features of 8 hypothyroid infants and children observed over long periods of inadequate and adequate therapy are discussed. Histories are presented in conjunction with critical roentgenograms to demonstrate: (a) peristence of retardation during inadequate treatment and (b) rapid progress in maturation with adequate therapy.

Periodic roentgenograms have been found to be a valuable guide to therapy in the hypothyroid infant and child. Inadequate treatment during the early critical months of life is associated with mental retardation as well as delayed skeletal maturation. Doses of desiccated thyroid or sodium l-thyroxine which are sufficient to reverse many clinical and metabolic signs of thyroid deficiency are often inadequate to produce normal bone maturation and continuing growth of the central nervous system. Adequate therapy begun at an early age promotes immediate and continuing skeletal development to normal levels. Some degree of mental retardation may persist.

Routine serial roentgenograms were obtained as follows: (a) posteroanterior of both hands; (b) anteroposterior of left elbow; (c) anteroposterior of left hip; (d) anteroposterior of left knee; and (e) lateral of left ankle. Roentgenograms of the hand and wrist were found to be of greatest value for diagnosis and evaluation of therapy. Interpretation of the films was based on the maturity indicators of the individual bones of the hand and the epiphyses as demonstrated by Gruelick and Pyle.—*A. W. Sommer, M.D.*

Cayler, Glen G., and Peterson, Carrell A. Infantile cortical hyperostosis. *A.M.A. Am.*

J. Dis. Child., Feb., 1956, *91*, 119–125. (From: Departments of Medicine and Radiology, Children's Hospital of the East Bay, Oakland, Calif.)

The roentgen finding, in young infants, of cortical subperiosteal new bone formation, commonly involving the mandible, usually associated with an extremely variable course of fever, swelling, and irritability was identified as a new syndrome by Caffey and Smythe about ten years ago. The course is usually benign and self-limited, although several deaths from intercurrent infections have occurred and there have been several instances of bony deformity. Thus far, all cultures and serologic tests for an infectious agent have been negative. Several cases have been observed at birth and a few even in utero. The roentgenographically demonstrated subperiosteal new bone formation is a nonspecific change, but when it involves the mandible of a young infant it is almost always due to infantile cortical hyperostosis.

Seventeen cases of infantile cortical hyperostosis are reported, 71 per cent of which were in girls. The average age of onset was fourteen weeks with a range of from four to thirty weeks. Approximately 70 per cent of the cases presented a chief complaint of swellings about the face. These swellings may be confused with parotitis, parotid tumor, orbital tumor, syphilis, facial trauma, lacrimal duct obstruction, facial cellulitis, scurvy, osteomyelitis, tuberculosis, Ludwig's angina, and familial fibrous swelling of the jaw. Differentiation is based on the roentgen findings of periosteal new bone formation along the mandible associated with onset within the first six months of life, adequate vitamin intake, no known history of trauma, absence of redness of the skin, no local adenopathy, negative serologic test for syphilis, and negative tuberculin skin test.

Therapy consisted of general supportive and symptomatic care, with the addition of blood transfusions in 5 cases. As far as it is known, no deformity occurred in any case.—*John F. Weigen, M.D.*

Zimmer, James F., Dahlin, David C., Pugh, David G., and Clagett, O. Theron. Fibrous dysplasia of bone: analysis of 15 cases of surgically verified costal fibrous dysplasia. *J. Thoracic Surg.*, April, 1956, *31*, 488–496. (From: The Mayo Foundation, Rochester, Minn.)

The authors report on their experience with 15 cases of fibrous dysplasia of the ribs. The patients ranged from eighteen to sixty-five years of age. Eight patients were women and 7 were men. Clinical signs and symptoms were few. Eight patients complained of chest discomfort, and 7 were asymptomatic. Ten patients had no physical findings, 2 had local tenderness, and 3 had noted some enlargement of the affected rib. A roentgenogram of the thorax revealed the lesion in each case. The lesions were found to be uniformally expanding central fibrous masses, ranging in size from 5 by 1 by 1 cm. to 19 by 6 by 5 cm. The expanded rib cortex showed irregular thinning. The internal surface of the cortex often exhibited erosions, the shadows of which appeared as septa in the roentgenograms. In many cases the diagnosis could be made from the roentgenogram with considerable certainty. In some cases, the lesion may not be differentiated roentgenologically from central cartilaginous tumors, myeloma, or eosinophilic granuloma.—*Paul C. Olfelt, M.D.*

Canepa, Giuseppe, and Seghini, Giuseppe. Osteocondrodistrofia dell'accrescimento. (Osteochondrodystrophy of growth.) *Arch. di chir. ortop. e di med.*, part I, Jan.-Feb., 1956, *21*, 26–69, part II, March-April, 1956, *21*, 79–127. (From: Istituti Ospedalieri di S. Corona in Pietra Ligure, Savona, Italy.)

After surveying previous attempts to classify the highly eponymic identification of developmental bone disturbances, and before describing 12 (8 of which reported for the first time) cases, the authors repeat the often expressed desire to unify the nomenclature of these syndromes.

The term *osteochondrodystrophy of growth* (suggested by Marziani) should include all the types; its pure, complete picture would be a combination of the *familial osseous dystrophy* of Morquio and of the *chondrodystrophy* of Brailsford. Incomplete, atypical, or late variants were the *endemical osteoarthritis deformans* of Kaschin-Beck, the *hereditary multiple epiphysitis* of Müller-Ribbing, the *spongious epiphyseal dystrophy* of Ghimus, the *multiple osteochondropathy* of Grudzinski, the *late chondrodystrophy* of Catel, and the *abortive chondrodystrophy* of Budde. Conversely, the *fruste chondrodystrophy* of Silfverskiold, the *familial pleonosteosis* of Léri, and the *dyschondro-osteosis* of Léri-Kajani-Well are considered transitional stages between the pure osteochondrodystrophic types and achondroplasia. Hurler's *gargoylism* could then be regarded as a late (worsened) Morquio-Brailsford type.

The literature contains descriptions of variants, which have received an eponymic label for minor differences. Wide acceptance of a generic term, correlated with a basic set of symptoms and objective findings would certainly facilitate the recognition of these cases. Moreover, many more of these patients will survive into adulthood than in previous years, and we will encounter them even outside of the territory of pediatric practice.—*E. R. N. Grigg, M.D.*

Harmston, Gordon J. Osteopathia condensans disseminata. *Radiology*, April, 1956, *66*, 556–

560. (Address: 110 North First, East, Logan, Utah.)

Osteopathia condensans disseminata is usually discovered incidentally during the process of obtaining roentgenograms for diagnosis. This condition is rare and the author adds 5 cases to the 81 previously reported in the world literature. The terminology used in reporting these cases has been confused, with terms such as: osteopoecilia, osteopoikilosis, osteitis condensans generalisata, osteosclerosis fragilitas generalisata, and osteopathia condensans disseminata having been employed. The last term is thought to be the most appropriate.

There are no symptoms associated with this condition and the etiology is unknown. In some of the series reported, a familial tendency has been found. The cases reported in this paper occurred in a father, 3 daughters, and a son, indicating a definite familial and hereditary predilection, and that the condition is transmitted through males.

Roentgenologically, rounded, oval, or elongated areas of increased density, from 0.2 to 1.0 cm. in diameter, are seen lying within the marrow spaces. If the lesions are oval the long axis lies parallel to the long axis of the bone or, in the flat bones and epiphyses, follows the general direction of the trabeculae; they tend to be localized at points of stress and motion. The bones most commonly affected are the pelvis, femurs, carpal and tarsal bones, while the skull, ribs and spine rarely show involvement which tends to be bilateral and more or less symmetrical. Serial roentgenographic studies over a period of years demonstrate evidence of slow change in these islands of bone. Some foci enlarge, some disappear entirely, and others make their appearence where none existed previously, indicating activity of osteoblasts and osteoclasts. The involved bones are all otherwise normal.

The differential diagnosis includes: osteoblastic metastatic cancer, tuberculosis of bone, diffuse osteomyelitis, and osteopetrosis.—*Donald N. Dysart, M.D.*

Stein, George N., Israel, Harold L., and Sones, Maurice. A roentgenographic study of skeletal lesions in sarcoidosis. *A.M.A. Arch. Int. Med.*, May, 1956, *97*, 532–536. (From: Departments of Radiology and Medicine of the Graduate Hospital and the Sarcoid Clinic of the Henry Phipps Institute of the University of Pennsylvania and from the Woman's Medical College of Pennsylvania, Philadelphia, Pa.)

Roentgenograms of the hands and feet of 81 patients proved to have sarcoidosis showed characteristic osseous lesions in 17.2 per cent.

The authors used the criteria of single or multiple, round or oval radiolucencies in the epiphyses surrounded by a zone of normal bone as the first type of involvement. This is the usual "punched-out" area. The second type of bone change is the diffuse type, in which the cortex of the bone is thin and the medullary portion widened and the trabecular pattern has a reticular appearance. This gives a honeycombed or lattice work configuration. The third type is the mutilating one, in which the lesions appear to coalesce and produce destruction of the bone. Slight generalized osteoporosis was not regarded as diagnostic. A moderate degree of osteoporosis or small changes in the trabecular pattern were regarded as suspicious, but these alterations were not included in the positive findings.

In another group of 94 sarcoidosis suspects, definite osseous lesions were found in only 3 per cent.

In one patient only were these lesions felt to have caused symptoms.

Complete bone surveys were made of 10 patients having lesions of both the hands and feet, and involvement of bones other than the hands and feet was quite rare.

Roentgenographic demonstration of lesions in the bones of the hands and feet provides valuable corroborative evidence for the diagnosis of sarcoidosis and should be routine in all patients in whom sarcoidosis is suspected.—*G. L. Sackett, Jr., M.D.*

Sherman, Robert S., and Soong, Kenneth Y. Ewing's sarcoma: Its roentgen classification and diagnosis. *Radiology*, April, 1956, *66*, 529–539. (Address: Dr. Sherman, Memorial Center, 444 E. 68th St., New York 21, N. Y.)

One-hundred and eleven cases of histologically proved Ewing's sarcoma from the files of Memorial Center (New York) were reviewed. They were selected on the basis of adequate roentgen study, dependability of histologic diagnosis, and clinical correlation. The results of a detailed study of the roentgen aspects are reported.

The group was composed of 77 males and 34 females. The age distribution was as follows: 29 cases up to the age of nine, 47 between the ages of ten and nineteen, 30 between twenty and thirty, and 5 above the age of thirty. The skeletal distribution was as follows: long bones 63 cases, ribs 16 cases, pelvis 18 cases, scapula 10 cases, and small bones 4 cases. No example of primary tumor in the skull, mandible or vertebra was encountered in this series. Metastases, however, were found in all parts of the skeleton.

For the purpose of detailed study the tumors were divided into 2 groups. On the basis of their skeletal location they were separated into those in the long bones and those in the remainder of the skeleton. Great variation was noted in the roentgen appearance of Ewing's sarcoma of the long bones, with only 29 of the 63 cases considered to present a classical picture. Most of the tumors in the central diaphyseal

location presented a classical picture. The typical roentgen features displayed in 23 tumors in this group were essentially as follows: mid-shaft location, long extent of the new growth, fusiform configuration, frequent involvement of soft parts, fine patchy integral pattern of bone destruction, ill-defined edges, and a parallel form of periosteal reaction. Some degree of cortical destruction was the rule.

The most characteristic feature of the lesions located in the cortical portion of the mid-shaft was cortical erosion of varying degree involving the outer surface while the inner surface remained intact. These tumors were invariably asymmetrical and there was a large extra-osseous soft-tissue component. The direction of growth seemed to be external to the bone rather than internal.

Tumors in a central metaphyseal location varied from the classic type in diversity of the internal pattern. Patchy bone destruction was always seen but sclerotic changes were not infrequent. Tumors in an eccentric metaphyseal location frequently simulated the mixed type of osteogenic sarcoma. Contrary to the general belief that the epiphysis is not affected in Ewing's sarcoma, 6 instances were encountered in which this part of the bone was invaded. There were no instances of clear-cut epiphyseal involvement alone. Ewing's sarcoma occurring in the flat bones offered no findings that could be depended on for roentgen identification.

Excellent roentgenograms, illustrating the various types of Ewing's sarcoma, are reproduced.—*A. W. Sommer, M.D.*

Jacqueline, F. (Aix-les-Bains, France.) Les coxarthroses. (Arthroses of the hip.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, Jan.-Feb., 1956, *37*, 16–30.

This report is based on an extensive experience with primary and secondary hip arthroses over many years. The severity of the arthrosis is due essentially to the amount of bone damage as indicated by condensation and poorly defined translucent areas adjacent to significant areas of destruction. Disturbances in the soft tissues are responsible for pain.

Primary arthrosis begins with an inferomedial narrowing leading to superolateral widening of the joint space. However, this finding should be interpreted with caution because it can be simulated when the film is exposed in internal rotation. Other early signs are sclerosis of the anterior and posterior horns, and of the inferomedial portion of the femoral head, osteophytes about the fovea, at the inferior part of the junction of head and neck of the femur and at the elongation of the base. The evolution of the primary disease is variable and depends on how quickly the head of the femur is raised and how badly it is positioned. For a long time, the bony change is limited to the head; it appears in the acetabulum only when the supporting surface is markedly reduced.

Frankly dislocated hips may show acetabular sclerosis which need not be considered a sign of arthrosis. Bone damage appears first in the acetabulum, followed by changes in the femoral head as the opposing surface becomes smaller. With protrusion of the hip, there is a diffuse sclerosis, which remains localized to the acetabulum.

The initial localization of the bone damage gives information regarding prognosis by establishing presence of significant malformations, adaptation to the new direction of the forces develops slowly and relatively satisfactorily. The arthrosis is associated with sclerosis and is well tolerated. When fibrosis of the capsule results in joint modifications, changes in the acetabulum appear.

The normal femoral head is more sensitive to inferomedial narrowing than to elevation. In patients over 45 or 50, this may result in circulatory changes. With a deep acetabulum, however, the arthrosis is likely to be benign.—*Marcy L. Sussman, M.D.*

Blood and Lymph System

McCallen, Albert, and Schaff, Burnett. Aneurysm of an anomalous right subclavian artery. *Radiology*, April, 1956, *66*, 561–563. (Address: Dr. McCallen, Veterans Administration Hospital, Coral Gables, Fla.)

An anomalous right subclavian artery arising as the fourth branch from the left side of the aortic arch is one of the most common anomalies of the great vessels. Felson, *et al*, and Coplemann have published excellent discussions of this condition. A search of the literature has, however, failed to reveal a report of a true aneurysm of an anomalous right subclavian artery. The authors have, therefore, presented a case report of a patient who had this condition. The diagnosis was made in this instance by the radiologist from roentgenographic and roentgenoscopic findings, and was confirmed by autopsy. At autopsy, the aneurysm was found to have developed secondary to atheromatous changes in the vessel wall.

The authors point out that aneurysm of an anomalous right subclavian artery is a condition which must be considered in differential diagnosis of superior mediastinal masses.—*A. Ralph Watson, M.D.*

Kaufman, Joseph J., Burke, Donald E., and Goodwin, Willard E. Abdominal venography in urological diagnosis. *J. Urol.*, Jan., 1956, *75*, 160–168. (From: Division of Urology, Veterans Administration Hospital and the University of California Medical Center, Los Angeles, Calif.)

The authors have performed vena cavagrams in 50 patients by either bilateral percutaneous femoral vein puncture, translumbar vena cava puncture or percutaneous femoral vein catheterization. Blood pressure cuffs are applied to the thighs and the Valsalva maneuver is employed during the roentgen exposure.

Untoward reactions consisted of a transient discomfort due to extravasation of contrast medium outside the femoral vein in 20 per cent of the patients, but the procedure is considered to be as safe as intravenous urography. Contraindications to the method are sensitivity to the contrast medium or the presence of active thrombophlebitis.

By this method the authors have been able to demonstrate invasion and compression of the inferior vena cava by renal or testicular neoplasms, retrocaval ureter, obstruction or displacement of the inferior vena cava by any retroperitoneal neoplasm. A preoperative venogram may be of value when surgery of the vena cava is contemplated.—*George W. Chamberlin, M.D.*

Stauffer, Herbert M., Durant, Thomas M., and Oppenheimer, M. J. Gas embolism; roentgenologic considerations, including the experimental use of carbon dioxide as an intracardiac contrast material. *Radiology*, May, 1956, *66*, *686*–692. (Address: H. M. Stauffer, Temple University Hospital, 3401 North Broad St., Philadelphia 40, Pa.)

The authors present an excellent discussion of gas embolism. This article is particularly timely, as the use of gas injection for contrast in diagnostic roentgenology is increasing. The danger of embolism is always inherent in such diagnostic procedures as well as in some therapeutic procedures in which entry of air into the circulation may occur.

It is pointed out that venous and arterial gas embolism produce their adverse effects by somewhat different mechanisms, and that the two forms of embolism present entirely different clinical pictures. In venous embolism, the air enters one of the systemic veins and produces its effect by an air trap in the outflow tract of the right ventricle and main pulmonary artery, and, when the patient is supine, by blockage of the small peripheral pulmonary arteries. The air in the right ventricle produces a loud churning sound which has been termed a "millwheel" murmur. In arterial embolism, the air enters the pulmonary venous channels and is carried to the superiorly located systemic arteries so that small quantities of air may produce serious blockage of coronary or cerebral vessels. Thus, neurologic manifestations or signs of coronary occlusion may develop.

As a result of experimental and clinical studies, a number of facts which have practical importance are presented. Since air and oxygen are shown to be dangerous if embolism occurs, a readily soluble gas such as carbon dioxide or nitrous oxide should be used whenever possible for diagnostic or therapeutic insufflation.

Should air embolism be suspected, the patient should be immediately placed in left decubitus position with the head dependent. This position tends to eliminate the air trap which may result from venous embolism, and may prevent arterial air embolism from reaching the cerebral circulation. Artificial respiration should be instituted at once, and if other measures fail, aspiration of air from the right atrium may be attempted.

The possibilities of carbon dioxide as a contrast medium for angiocardiography are discussed, since it was found that rapid intravenous injection of large amounts of this gas in dogs produced only minor, transient physiologic alterations. Two infants tolerated similar doses well.—*A. Ralph Watson, M.D.*

General

Abel, Martin S. Advantages and limitations of the 0.3-mm. focal spot tube for magnification and other technics. *Radiology*, May, 1956, *66*, 747–752. (Address: 655 Sutter St., San Francisco 2, Calif.)

Focal spot size affects roentgenographic sharpness because of its direct effect on penumbra size. Magnification of both the object and penumbra is increased by either a decrease in focal spot-object distance or an increase in object-film distance, but the penumbra size increases more rapidly than the image size. With an ultra-fine focal spot, the relative penumbra size is smaller with magnification than with a large focal spot without magnification. Of course, the relative penumbra size will be smallest with the least amount of magnification, regardless of focal spot size. In addition to over-all magnification, differential magnification between superimposed parts of the body is often useful, because of the perspective gained.

The author has found that maximum roentgenographic detail is obtained with magnifications of 1½ to 2. The upper limit of practical usefulness of magnification is a little over 3, because beyond this fine bony detail is lost.

Clinically the use of a small focal spot tube (0.3 mm.) has been found to be of value in the following situations: (1) studies without magnification of parts anatomically considerably removed from the film; (2) portable examinations, or other such situations where routine technique is impossible; (3) magnification studies where enlargement or increased detail is desired; (4) situations where differential magnification would be of aid in separating superimposed structures in planes of the body quite close together. It is sometimes helpful to purposely exaggerate the magnification to the extent that structures farthest

from the film are obscured, making closer structures stand out with greater clarity; (5) laminagraphy; and (6) fluoroscopy, in order to decrease the penumbra effect which is prominent because of the short focal spot-object distance.

With the use of a small focal spot, the operating milliamperage of 12–15 ma. is a disadvantage, especially for thick parts. In order to minimize the secondary radiation and not unreasonably prolong the exposure time, the author uses the highest practical kilovoltage, the smallest possible cone, and a focal spot-patient and patient-film distance of 20 to 24 inches.

Magnification techniques have their limitations. Complete immobilization is necessary because of the long exposure time, and so these techniques are employed mainly for the bones and joints. Usually the techniques are limited to localized views because of the extreme coning needed for thick parts. Sometimes magnification in undesirable because evaluation of a lesion depends more upon over-all variations in density and structure. As the author so aptly says "with magnification, the forest may not be seen for the trees."—*Charles L. Stevenson, M.D.*

Lasser, Elliott C., and Nowak, Edward L. Multiple simultaneous body-section radiography. *Radiology*, April, 1956, *66*, 577–581. (Address: Dr. Lasser, Department of Diagnostic Radiology, Roswell Park Memorial Institute, Buffalo 3, N. Y.)

The development of a method for multiple simultaneous laminagraphy dates back to the work of Ziedes des Plantes, in 1933. In spite of its advantages over conventional laminagraphy, it has been used only sporadically in this country. It saves time and reduces the radiation exposure for the patient.

By utilizing a vertical stack of films separated by suitable spacings, instead of a single film, multiple laminagraphic images can be recorded by a single tube-film movement, with a different plane of image clarity on each film in the stack. The separation of the body planes is not identical with the separation of the films, but depends upon the tube-film distance and the height of the fulcrum. The greater the fulcrum height, the smaller the spacing between body planes.

In order to compensate for the difference in radiation flux reaching the different film levels (due to absorption from higher films, screens, etc., and the fall-off in accordance with the inverse-square law), suitable combinations of various speeds of intensifying screens and films are used with controlled development of individual films.

The authors have devised a special cassette, which will hold five 10 by 12 inch films separated by $\frac{1}{4}$ inch lucite spacings. The details of its construction and the combination of the intensifying screens and films employed at various levels are given. In order to use a cassette this thick, the Bucky apparatus must be modified, and the details of this are provided.—*Charles L. Stevenson, M.D.*

Wang C. C., and Robbins, Laurence L. Amyloid disease. *Radiology*, April, 1956, *66*, 489–501. (Address: Dr. Wang, Massachusetts General Hospital, Boston 14, Mass.)

In amyloid disease there is deposition of a starch-like material called amyloid in the lungs, heart, blood vessels, and less frequently, in the spleen and gastrointestinal tract. Primary amyloidosis is not associated with chronic sepsis, nor with any other known etiologic factor. Secondary amyloidosis, which is more common, usually follows long standing infections and tissue-destroying processes such as rheumatoid arthritis, bronchiectasis, tuberculosis and malignant tumors. Deposits are found in the liver, spleen, kidneys and adrenal glands.

Clinically, the signs and symptoms vary, depending upon the system involved. When the kidneys are infiltrated the findings are similar to those of the nephrotic syndrome. The liver or spleen may be palpable if involved. Hematemesis, epigastric distress or pyloric obstruction may be seen with stomach involvement. When the small intestine is infiltrated, chronic diarrhea is a frequent manifestation. Decompensation is seen late when the heart is involved. When infiltration in the lungs becomes massive, progressive dyspnea develops.

In a study of the autopsy reports from Massachusetts General Hospital in Boston, a total of 29 cases of this disease was found. In 19 of this group, roentgenograms were available for study and this material constitutes the basis of the present investigation.

The roentgenologic manifestations vary depending upon the amount and location of infiltration in the particular organs affected. Hepatomegaly, splenomegaly and enlargement of the kidneys may be seen when these organs are involved. Functional impairment without noticeable distortion of the collecting system accompanies the kidney enlargement.

Early in the lung involvement increased prominence of the bronchovascular markings with inconspicuous stippling, when the blood vessels are chiefly involved, is seen in the peripheral lung fields. In the late stages, when amyloid is deposited in the interstitial tissues, the appearance is that of a miliary process such as is seen in miliary tuberculosis, silicosis, sarcoidosis, and granulomatous disease. In severe cases the findings mimic lymphatic spread of a malignant growth. Hilar lymphadenopathy is seen when these nodes are involved.

Gross heart enlargement followed by congestive failure is observed when massive infiltration of the heart is present.

The gastric manifestations consist of ulceration, tumor formation, hypertrophy of gastric folds and

pyloric stenosis. Increased widening and separation of the mucosal folds, with some dilatation of the lumen, is seen when the small intestine is involved. Impairment of intestinal motility and a roentgenographic appearance of ileus is evident when the involvement is severe.

In conclusion the authors state that these roentgen changes in the various organs are not pathognomonic of amyloid disease, but their correlation with the clinical data and laboratory findings may lead to the correct diagnosis.

Seven case studies are presented and numerous roentgenograms are reproduced illustrating these findings.—*Donald N. Dysart, M.D.*

Gannon, William E., and Greenfield, Henry. Fibrin body in an old abscess cavity, stimulating a new growth. *Radiology*, April, 1956, *66*, 564–566. (Address: Dr. Gannon, 855 Ocean Avenue, Brooklyn, N. Y.)

Fleischner was the first to report the occurrence of opaque fibrin bodies in the pleural cavity, and their etiology and significance has been elaborated by many authors. They have also been found in the peritoneal cavity, pericardial sac, and the renal pelvis. In the chest, they are usually associated with long standing hydropneumothorax, and the authors suggest that they arise from hemorrhage rather than from fibrin flakes in the pleural exudate. Gradually they may decrease in size, or entirely disappear.

The presence of a fibrin body in an old epithelialized abscess cavity in an elderly white male is reported. Preoperatively its appearance suggested a new growth.—*Charles L. Stevenson, M.D.*

RADIATION THERAPY

Maisin, J., Maisin, H., and Keusters, J. Utilisation des super-voltages et des grandes masses de radioelements dans le traitement du cancer en general. (The use of supervoltages and of large masses of radio-elements in the treatment of cancer in general.) *J. belge de radiol.*, 1956, *39*, 88–126. (From: Institut du Cancer, Université Catholique de Louvain, Louvain, Belgium.)

After general considerations regarding super- and megavoltage radiation therapy, the authors relate their experience with the 2 radium sources—50 and 15 grams—donated by the Union Minière (the company which produces radium in the Belgian Congo).

The 50 gram source, supported by a 20×6 cm. plate, is movable within a cylindrical space, carved in a solid 65×50×65 cm. lead block. When not in use, the source is lowered and a 25 cm. thick lead screen, weighing two tons, covers the opening of the cylindrical space. The patient is positioned on a special table (a bed with aluminum springs) located above the radium container. In this arrangement the beam is vertical, and directed upwards. The table contains a special mechanism which allows for linear and pendular motion, provided to give both arc therapy and cyclotherapy. The output is 345 r/h. at 35 cm. without added filter, while 10 mm. Al (used in treatment of patients) will reduce it to 258 r/h. Depth doses were determined with a Victoreen chamber in a rice phantom: with a 22×6 cm. field, at 35 cm. distance, for 100 per cent on the surface there will be 75 per cent at 5 cm. depth, 48 per cent at 10 cm., 29 per cent at 15 cm., and 12 per cent at 20 cm.

The 15 gram source, encased in a lead head, is surrounded by 15 cm. of lead in the rest position. This head is very flexibly mounted on a tube stand, allowing angulation up to 300°. A backpointer is also used with this source. Several localizers (collimators) are employed: 8.5×8.5, 8.5×5, 5×5, and 2×2 cm., the lengths being 6, 12, 15, and 18 cm. The distal orifice of every collimator contains a filter of $\frac{1}{4}$ mm. Cu +1.0 mm. Al, giving at 6 cm. an output of 1530 r/h., at 12 cm. 540 r/h., and at 18 cm. 210 r/h. The depth dose for the 12 cm. distance is at 2 cm. 72 per cent, at 4 cm. 53 per cent, at 10 cm. 25 per cent, and at 20 cm. 8 per cent. This 15 gram source is used mostly for the treatment of larynx and lung tumors, but no results are quoted because it was acquired too recently (1953).

The large source was a 30 gram source from 1950 to 1951, when it became a 47 gram source, while the other 3 grams were added in 1952. The tumors treated were mostly of moderate radiosensitivity (carcinoma of cervix, esophagus, bladder, rectum, prostate, nasopharynx). Comments are offered only regarding the carcinoma of cervix cases.

A comparison is made with their own statistics of 1,256 patients with carcinoma of the cervix, treated between 1924 and 1948, with combined external roentgen therapy (four opposing pelvic ports, 400 kv., 1,000–1,500 r in air to each port) and intracavitary radium (3,000–5,000 mg. hr. to applicators is os and colpostats). There were five-year survivals of 76.7 per in Stage I, 50 per cent in Stage II, 29.3 per cent in Stage III, and 3.8 per cent in Stage IV carcinomas.

Telecurietherapy was used with best results in association with the pendular motion therapy (cyclotherapy). It delivers 100 per cent of the skin dose to a cylinder of 10 cm. diameter and 20 cm. length. One can deliver 6,000–7,000 gamma r to the cervix in this way. Since there is, however, 200 r/h. entrance skin dose and about 30 r/hr. exit skin dose, a fairly strong reaction is usually seen. The authors emphasize that the rectal and vesical disturbances are not very severe, nor is there a significant systemic reaction present, although a transient depression of the blood count occurs frequently. Forty-one Stage II, and 56 Stage III patients were treated either with telecurietherapy alone, or combined with

local radium application. Provisional estimates, obtained from up to four year observations, seem to indicate that Stage II carcinoma of the cervix can be controlled by telecurietherapy alone, while for Stage III external roentgen therapy combined with intracavitary radium appears to be superior to telecurietherapy, whether alone or combined with local radium. Adequate follow-up of a statistically more significant number of patients will be required for the final evaluation of the method.—*E. R. N. Grigg, M.D.*

BAILY, NORMAN A., and BEYER, NORMAN S. Beam quality determinations for a 2-Mvp resonance type x-ray generator. *Radiology*, April, 1956, *66*, 572–576. (From: Roswell Park Memorial Institute, Buffalo, N. Y.)

This study was undertaken in order to determine the effects of introducing large amounts of absorbing material in the path of high energy electromagnetic radiation. The results were expressed in terms of the half-value layer of lead at different distances from the target after a variable thickness of initial filtration was introduced and varying thicknesses of lead absorbers were placed at different positions.

It was found that although there is a large increase in the measured half-value layer with the absorbing material 95 cm. from the target rather than at 5.0 cm., a slight but significant decrease from the 5.0 cm. value is observed when the absorbers are at 53.5 cm. from the target. This is thought to be due to multiple scattering by the lead diaphragms and shutters. The soft radiation, which would be scattered out of the beam if these were not present, is scattered back into the sensitive measuring volume. Since this multiple scattered radiation is of reduced energy, it will undergo large-angle scatterings upon further passage through lead, and will not traverse the active volume, thus making the absorbers appear more effective than those placed 5.0 cm. from the ionization chamber.

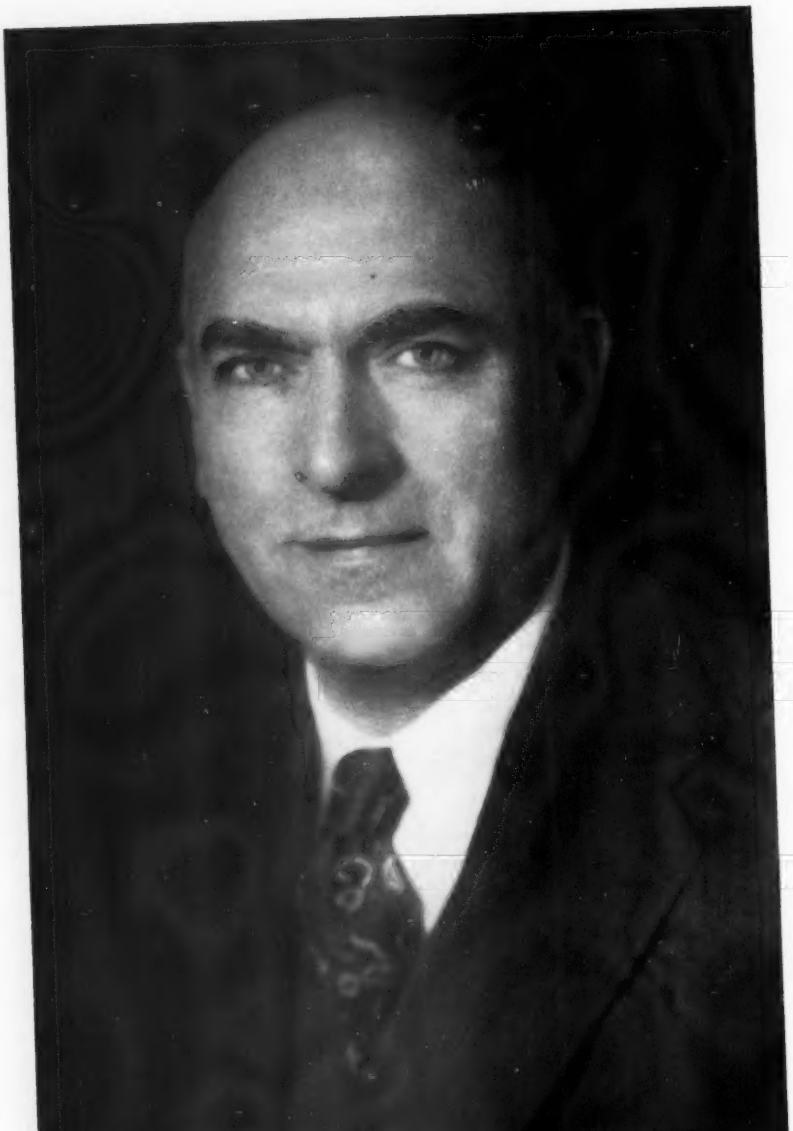
These studies show that the different half-value layers obtained are a function of the position of the absorbers placed in the high-energy roentgen-ray beam. The physical layout used in obtaining these values is illustrated, as are the transmission curves drawn on the basis of these data.—*Donald N. Dysart, M.D.*

TAVERAS, JUAN M., MOUNT, LESTER A., and WOOD, ERNEST H. The value of radiation therapy in the management of glioma of the optic nerves and chiasm. *Radiology*, April, 1956, *66*, 518–528. (Address: Dr. Taveras, Neurological Institute, 622 West 168th St., New York 32, N. Y.)

The case histories of 34 patients with primary glioma of the optic nerves and chiasm are reported in summary, and the use of radiation therapy and surgery in their management is evaluated. In the past, emphasis has been placed upon surgery, but the results of treatment in this series of patients show that roentgen irradiation is the procedure of choice. In the majority of instances in this series, surgery was limited to diagnostic craniotomy in obscure cases or procedures to relieve the obstruction of cerebrospinal fluid circulation which occurred in some patients. Tumors limited to the orbital cavity, as evidenced by absence of enlargement of an optic canal, were treated successfully by intra-orbital surgical removal. All other patients should be treated by irradiation.

Excluding all known living patients in whom the diagnosis of optic glioma was made during the last five years, 22 patients remain eligible for assessment. Three of the 7 deceased patients in this group died in the immediate postoperative period, having received no radiation therapy. Fifteen of the remaining 19 patients survived more than five years after irradiation was begun. Among the numerous beneficial results, the effect upon vision was outstanding. Restoration of lost vision or arrest of the advancing visual deficit occurred following treatment and persisted five years in 8 patients. In the other living patients who have been followed more than five years there has been a slow advancement of visual impairment, but no evidence of extension of the tumor beyond the optic nerve and chiasm and the general health has remained good.

Patients treated before 1950 received 3 courses of irradiation at intervals of six weeks with an average tumor dose of 1,200 r in each treatment series. In recent years most patients have received a single protracted series of roentgen treatments. At the present time the basic minimum dose for adults is considered to be 4,000 r to the tumor, given in twenty-eight days. A critical review of a few cases in this series seems to justify the trend toward more intensive irradiation.—*Harold C. Hamilton, M.D.*



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LLOYD F. CRAVER
Janeway Lecturer, 1956